"A STUDY ON CLINICOMICROBIOLOGICAL PATTERN IN CHRONICDIABETIC ULCER FOOT PATIENTS "

A DISSERTATION SUBMITTED TO

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In partial fulfilment of the regulations for the award of the degree of

M.S. GENERAL SURGERY – BRANCH I

DEPARTMENT OF GENERAL SURGERY

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

MAY 2019

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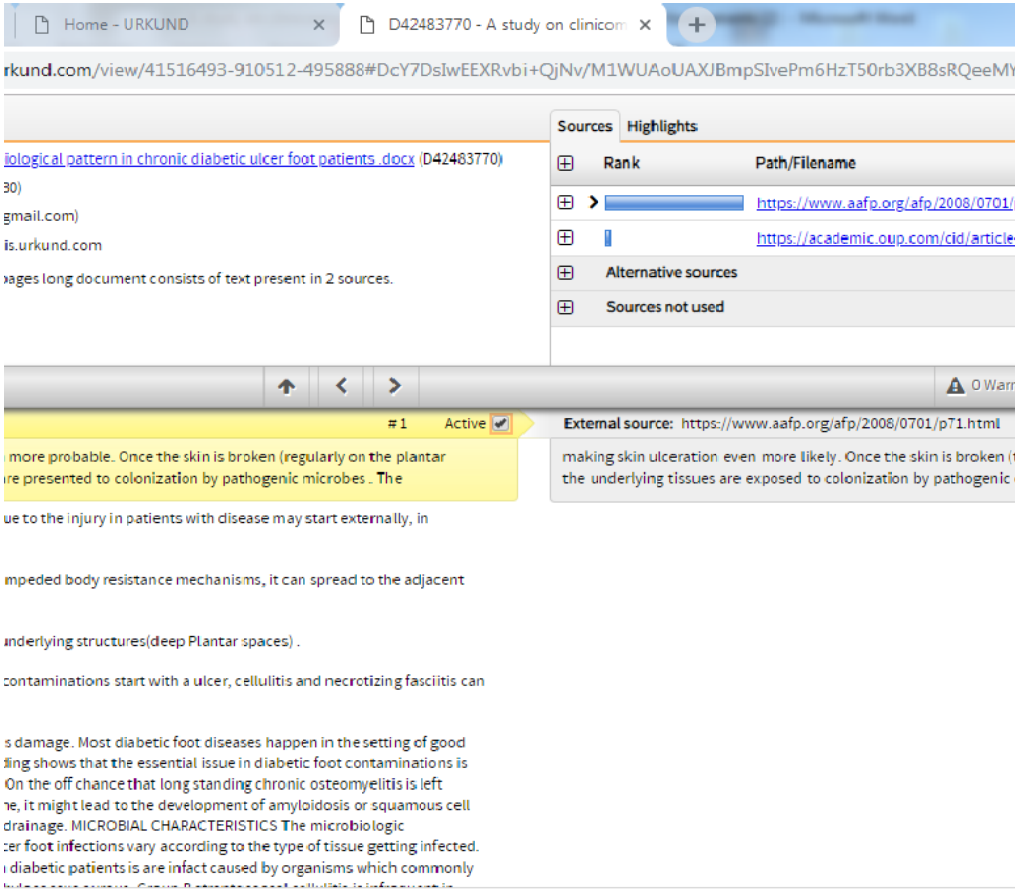
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DECLARATION

I solemnly declare that the dissertation titled "**A STUDY ON CLINICOMICROBIOLOGICAL PATTERN IN CHRONIC DIABETIC ULCER FOOT PATIENTS"** was done by me from 2016 onwards under the guidance and supervision of **DR. V. LEKSHMINARAYANI , M.S , D.G.O.**

This dissertation is submitted to the TamilnaduDr. M.G.R Medical University towards the partial fulfilment of the requirement for the award of M.S Degree in General Surgery (Branch I).

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DATE: SIGNATURE OF THECANDIDATE

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INTRODUCTION

Diabetes Mellitusis one of the commest metabolic health disorder in which there is increase in blood glucose levels over a long duration. WHO describes as" Diabetes mellitus is a chronic disease caused by inherited and/or acquired deficiency in production of insulin by the pancreas, or bythe ineffectiveness of the insulin produced. Such a deficiency results in increased concentrations of glucose in the blood, which in turn damage many of the body's systems, in particular the blood vessels and nerve".

There are 453 million people in the world living with diabetes as on April 2018. The global prevalence of diabetes has grown from 4.9% in 1980 to 10.5% in 2017. Whereas in India it is estimated that currently there are 72.3 million people are affected by the disease with an overall prevalence is about 17.7%. The prevalence of diabetes in Tamilnadu is 9.7% .prime reasons for this increasing trend of diabetes is due to increased age of the population, unhealthy diets, obesity and sedentary lifestyles.Worldwide around2.4 million people die due to the diabetes or its related causes.Diabetes leads to a number of serious complications in which, Diabetic foot ulcer (DFU) is a major medical, social, and economic problems and is the leading cause of hospitalization for patients. In addition it also caused increased mortality .Chances of

developing foot ulcer People with diabetes is found to be around28%. The annual cost of expenditure for the treatment of diabetes is vastly expensive; it was $323 billion in 2016 .improper foot care in chronic diabetes leads to ulcer which is further complicated by wound infections. Diabetic ulcer initially begins as a superficialwound and in the long run leads to dreaded complications for example gangrene and amputations .*Staphylococcus* aureus,*Escherichia coli*, Pseudomonas speciesProteus species, and Enterococci., are reported as common organism isolated from patients with diabetic foot infections . If not treated on time they culminate in amputation. Due to delayed antibiotic treatment at least one in five patientsgo for a lower extremity amputation. Characteristically the rates of amputation in patients diagnosed with diabetes are 10 to 20 times those of non-diabetic populations.Coexistence of foot ulcer and foot ischemia ensuesAmputation even more when. Other hazard factors include, low transcutaneous oxygen ,peripheral vascular disease, age, smoking and depriveddiabetic control. The main pathophysiology of foot infection inpatients with diabetes is quite composite which is owing to host-related conflicts and pathogen related factors. Host related conflictsinclude ,neuropathy ,arteriopathy, ,immunopathy nephropathy etc. And the pathogen related factors comprisesmicrobial load ,virulenceand antibiotic-resistance. Distorted foot develops an ulcer when trauma interrupts the defending skin cover. The underlying hypodermic

tissues quickly become inhabited with bacterium that cause infection. Infection typically begins as native response manifested by classic signs and symptoms of inflammation. Infection spreads to deeper tissues when not brought under control.Many patients, particularly with vasculopathy orperipheral neuropathy, these symptoms and signs is also diminished resulting in advocate the infection by the secondary findings like foul odor, friable or stained granulation. Micro-organisms harborthe deep dermal tissues of all chronic wounds, however the exactinterface between microbes in the wounds and compromised healing is mysterious. With regard to antibiotic treatment, there is a deficiency of evidence regarding its efficiency, ideal regimens or clinical indications for the treatment.Regardless of this lack of evidence, antibiotics are habitually a cornerstonein the treatment of chronic wounds and these cases receive ominously more antibiotic prescriptions (both systemic and topical) than age and sex-matched patients. Recentstrategies for antibiotics prescribing for such wounds are repeatedly based on expert estimation rather than scientific fact and may present difficulties in understanding and execution to the clinician. The increasing prevalenceof antibiotic resistance althoughis widely recognized, the associations between antibiotic resistance, justifications for antibiotic therapy are yet to be resoluted.

Appropriatecontrolling of these infections requires suitable antibiotic choice following antimicrobial vulnerability results. Additional morbidity and mortality iscaused by infection with multidrug resistant organisms (MDROs) which also may increase the duration of treatment. Although increasing antimicrobial resistance is aimportant problem in India, there is a scarcity for data on the occurrence of multidrug resistant organisms (MDROs) infection and consequence of such infection among DFU. Hence there arises the necessity to evaluate the infecting pathogens on a routine manner.

This study is done in the resolution to assess the clinical and the Microbial features of diabetic foot infection in the patients in and around Coimbatore, Tamilnadu. Thus it act as pre-emptivemethodology to report susceptibility pattern of pathogens and will help the clinician to use apt antibiotics.

AIMS AND OBJECTIVES

- The main aim and objectives for this study is to analyse the types of Bacteria causing wound infections in Diabetic patients in our hospital and also todetermine the antimicrobial susceptibility pattern of the frequently isolated Bacteria. Also to perceive the extended spectrum Beta-lactamase creators among Gram Negative Bacteria.
- To frame and device an emperical hospital antimicrobial strategy for patients with diabetic ulcers.
- And to guide the treatingclinician with early bacteriological diagnosis and appropriate anti-microbial selection which can retrieve the organ from amputation which inturndecrease the morbidity and mortality.

REVIEW OF LITERATURE

Diabetes mellitus is a cluster of metabolic diseases described as chronic hyperglycemia subsequent toeither inadequate insulin production, reduced tissue sensitivity to insulin or both. Long standing increase in blood glucose levels principalsthe onset of numerous diabetic complications particularlyperipheral vascular disease ,peripheral neuropathy, , increased susceptabilty tolocal and genralised infection and impaired wound healing. Diabetic ulcer foot may be described asaassembly of disordersby which ischaemia ,neuropathyand infection leading on to tissue breakdown which subsequently resulting in increased morbidity and ultimately amputation.

This review of literature will show light upon the nature and epidemiology of diabetic ulcer foot and its prevention and management, with an emphasis on studies from India . The review will be demonstrated with local experience of instituting a diabetic ulcer foot service at Coimbatore medical college Hospital in Tamilnadu.

DIABETES MELLITUS

Definition

"Diabetes mellitus is characterized by chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism resulting from in insulin secretion, insulin action, orboth".

Depending on the etiology of the DM, factors contributing to may include:

- Reduced insulin secretion
- Decreased glucose utilization
- Increased glucose production

or

"Level' of glycaemia at which diabetes specific complications occur than on deviations frorn population based mean" .

EPIDEMIOLOGY OF THE DIABETIC FOOT SYNDROME

- A quarter of the diabetic population is at increased risk Of foot injuries as a result of the presence of diabedc neuropathy or an arterial circulatory disorder. Every year 3 to 7% Of diabetics suffer a foot lesion for the first time.
- Foot ulcers occur in approximately 15% Of people with diabetes which accounts for 25% Of an admissions With hospital stay

being longer than the stay for other causes and the risk of amputation is 15 to 40 times greater in diabetics that in others.

- Diabetic foot ulcers account for more than 50% of non traumatic amputations and are associated with high ntes of moftality. Re amputation and contralateral limb amputation.
- India has 30 million diabetics at present and in the 2025 India is predicted to have 57 million diabetics.

INCIDENCE IN INDIA

Foot ulcer	:	1-5%
Toe amputation	:	2.9%
Below knee amputation	:	1.7%
Prevalence Of diabetic foot in India	:	5.6-10.5%

SOCIO - ECONOMIC IMPACT OF THE DIABET1C FOOT SYNDROME

Overall, the costs generated by are about three times as high as those produced by non — diabetics.Foot complicationsconstitute a major proportion of these.

With primary healing, about 30 % of the total cost derives from hospitalization, but where amputation is required this figure is 65% to

80% die avenge healing duration for diabetic foot lesions is about four months.

Ten percent of all lesions persist for more than one year, which incurs further costs for outpatient care. Fifteen percent of all foot ulcers in diabetics do not heal before the patient's death.

Most important cost item is namely the "cost" to the patient themselves in terms of the emotional trauma suffered and the loss of qualityof life and independence.

CIASSIFICATION

Type I

Type pathology

1A : Autoimmune beta cell destruction leading to insulin deficiency

1B :Develop insulin deficiency byunknown mechanism causing destructive process of beta cells Lack immunologic markers

Type II

It is a heterogeneous group of disorders characterized by

- Impaired insulin secretion
- Variable degree of insulin resistance
- Increased glucose production

Distinct genetic & metabolic defects in insulin action & or secretion give rise to common phenotype of hyperglycemia in type — 2 DM

Type — 2 DM is preceded by a period of abnormal glucose homeostasis classified as

- Impaired Fasting glucose (IFG)
- Impaired glucose tolerance (IGT)

DIAGNOSIS

The National Diabetic Date Group &World Health Organization have issued a diagnostic criteria for DM-2 based on the following facts:

- RBS> 200 mgs / dl or> 11.1 m mol / L with symptoms Of DM

(polyuria, polydipsia, weight loss)

- FBS>126 mgs /dl or >7.0 m mol / L
- 2 hr plasma glucose (during Oral GTT) > 200 mgs / dl or >11.1 m mol / L (Not recommended as a part of routine screening)

TABLE 1: DIAGNOSIS OF DIABETES MELLITUS

Terms	Definition
Random blood glucose (RBS)	Blood glucose levels without regard to time since last meal
Fasting blood glucose (FBS)	Blood glucose levels when there is no caloric intake for past 8 hours
2 hour plasma glucose (During Oral GTT)	The test should be performed using a glucose load containing the equivalent of 75 grams anhydrous glucose dissolved in water

Type 2 DM

Etio — pathogenesis

- Insulin resistance
- Abnormal insulin secretion

Most studies support the view that insulin resistance precedes insulin secretory defects and that diabees develops only if insulin secretion becomes inadequate.

Genetic considerations

Type 2 DM has a Strong genetic component

- The concordance of type 2 DM in identical twins is between 70 and 90%
- Individuals with a parent with type 2 DM have an increased of diabetes; if both parent have type 2 DM, the risk approaches 40%
- Insulin resistance, as demonstrated by reduced glucose utilization in skeletal muscle, is present in many non diabetic, first- degree relatives of individuals with type 2 DM
- Major genes that predispose to this disorder have yet to be identified, but it is clear that the disease is polygenic and multifactor. The gene for the protease, calpain 10, is associated with type 2 DM in Hispanic and some other populations
- Various genetic loci contribute to susceptibility, and environmental factors (such as nutrition and physical activity) further modulate phenotypic expression of the disease. However, definition of the genetic susceptibility remains a challenge because the genetic defect in insulin secretion or action may not manifest itself unless an environmental event or another genetic defect, such as is superimposed.

- Mutations of molecules involved in insulin (e.g,, die iruulinrceptor and enzymes involved in glucose homeostasis) account for a very small fraction of type 2 DM.

Pathophysiology

Type 2 DM is characterized by three pathophysiologic abnormalities.

- Impaired insulin resistance
- Peripheral insulin resisancc,
- Excessive hepatic glucose production

TABLE : 2 Pathophysiology of type Il diabetes mellitus

Peripheral insulin resistance or excessive hepatic glucose production

Pancreatic beta cells compensate by increasing insulin output

Progressive insulin resistance and compensatory hyperinsulinemia progress as the disease progress

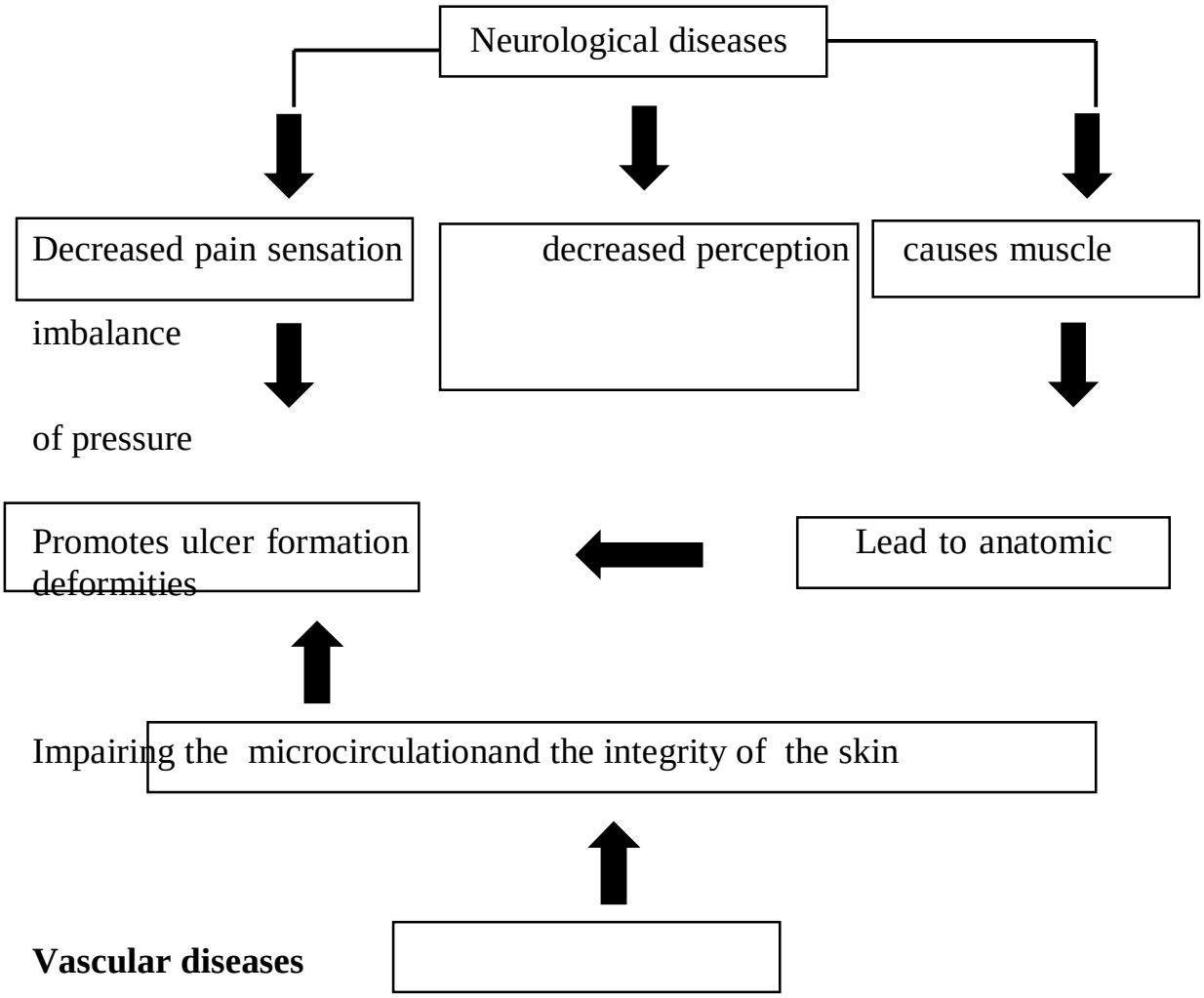
The pancreatic islets cells in certain individuals unable to sustain the hyperinsulinemic state

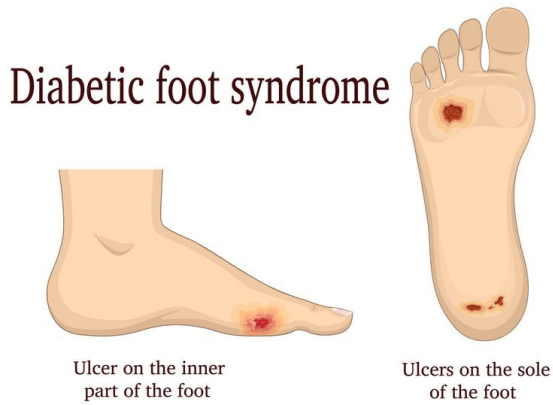
Result : IGT , characterized by elevations in post - prandial glucose. Decreased peripheral glucose usage results in post prandial hyperglycemia

A further decline in insulin secretion and an increase in hepatic glucose production lead to overt diabetes with fasting hyperglycemia .Increased hepatic glucose output predominantly accounts for increased FPG levels

Ultimately, beta cell failure may ensue.

TABLE : 3 Pathogenesis of Diabetic Foot





Physical signs resulting from diabetic neuropathy

The physical examination may reveal several abnormalities that result from such as

- claw toes
- Charcot arthropathy (also called diabetic neuropathic anthropopathy)

Vascular changes in diabetes

Atherosclerosis : chronic inflammatory process that can be converted into acute clinical events by plague rupture.

Development of atherosclerosis is accelerated in DM leading to oncreased morbidity and mortality.all the large vessels are involved in this process and clinical manifestations are apparent as a result of atherosclerotic narrowing and thrombosis of coronary,cerebral and leg vessels.

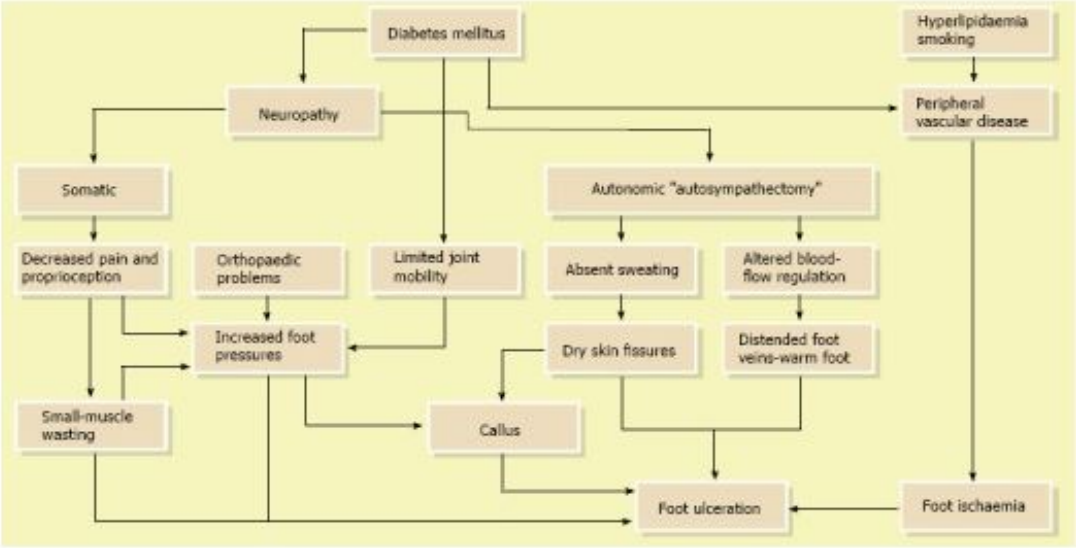


TABLE :4PATHOGENESIS OF DIABETIC ULCERS

Foot pressure abnormalities in diabetic foot

Diabetes mellitus

↓

Neuropathy & limited joint mobility

↓

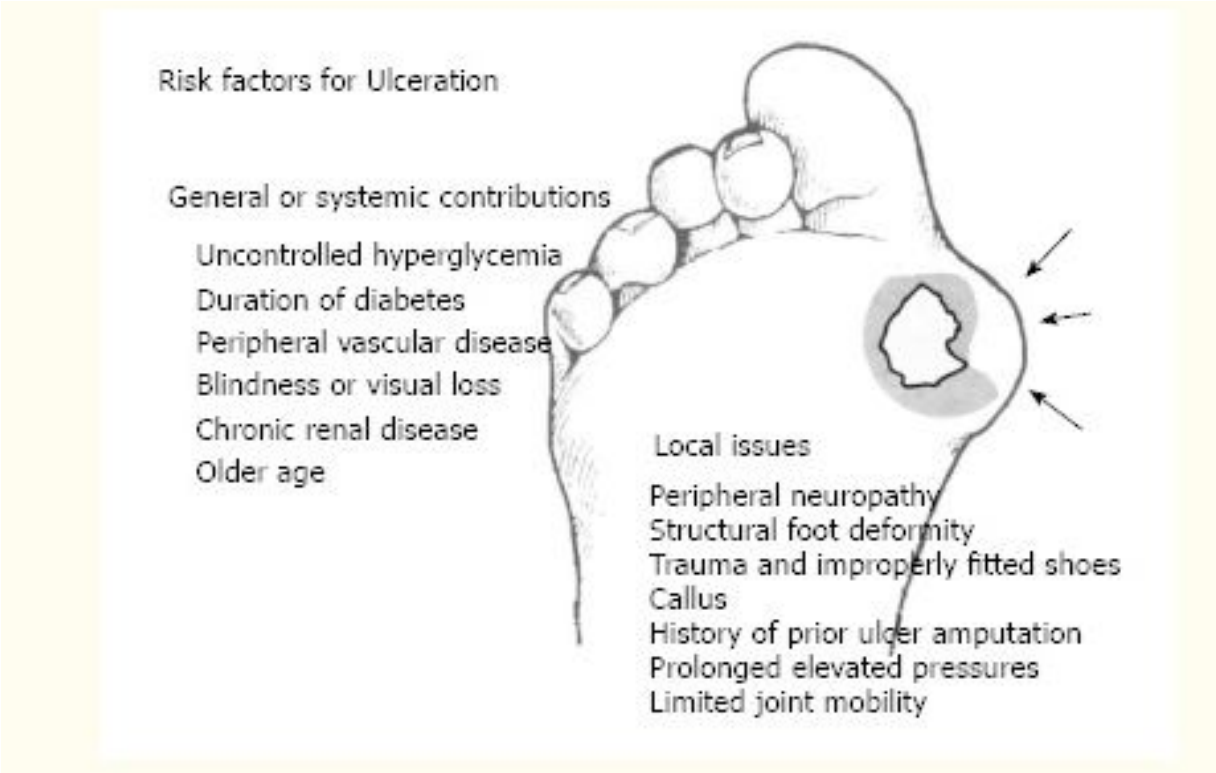
Atrophy Of intrinsic muscles & clawing

↓

Pressure transfer from heal & toes to metatarsal heads

↓

No redistribution of pressure



Predisposing Factors for Ulceration

1. Limited joint mobility

2. Peripheral neuropathy

3. High plantar pressure

4. Vascular diseases

TABLE 5 :CLASSIFICATION OF DIABETIC FOOT

ThcMeggitt - Wagner classification is the most well - known and validated system for foot- ulcers

Grade	**Description**
Grade 0	Symptoms like pain in the foot only is present
Grade I	Presence of superficial ulcer
Grade 2	Presence of deep ulcer
Grade 3	Ulcer involving bone
Grade 4	Gangrene of toes or forefoot.
Grade 5	Extensive gangrene involving entire foot

ULCERS

Diabetic ulcers are classified clinically as

- Neuropathic
- Ischemic ulcers
- Neuro ischemic ulcers

NEUROPATHIC ULCERS

Develop at areas of high plantar pressure. (metatarsal heads, plantar aspect of the great toe, heel or over bony prominences in a charcotfoot). Neuropathy is present in about 85-90% Of foot ulcers in diabetic patients. Are painless, unless they arecomplicated by infection.

There is callus formation at the borders of the ulcer. Its base is red, with a healthy granular appearance. On examination evidence of peripheral Neuropathy (hypoesthesia for complete loss of sensation of light touch, pain, temperature and vibration, absence of Achilles tendon reflexes, abnormal vibration threshold, often above 25v,atrophy of the small muscles of the feet, dry skin and distended dorsal foot veins) is present. However the pattern of sensory loss may vary considerably from patient to patient.

The foot has normal temperature of may be warm. Peripheral pulsesare present and ankle brachial pressure index in (n) or above 1.3.

ISCHEMIC ULCERS

Ischemia is a major factor in 38-52% of cases of foot ulcers. These ulcers develop on the borders or the dorsal aspect of the feet and toes or between toes. They are usually painful.

NEURO-ISCHEMIC ULCERS (MIXED ETIOLOGY ULCERS)

Neuro ischemic ulcers hase a mixed etiology neuropathy and ischemia.Despite the apparent signs of adequate perfusion, the foot is vulnerable to local "Micro Vascular" gangrene, will heal poorly and slowly, and will be less able to resist infection.

DIABETIC FOOT INFECTIONS

EPIDEMIOLOGY

Universally, diabetic foot diseases are the most well-known skeletal and tantalizing tissue contaminations in patients with diabetes. The occurrence of diabetic foot contaminations is like that of diabetes in different ethnic congregations and elderly patients are all the more ordinarily influenced. There are no critical contrasts between the genders. In USA occurrence of Amputation was 40,000 every year (Lancet, 2005) and in India the Incidence rate is @ 80,000 to one lakh removals for every year (2010 information of Vascular Society of India) which can be tip of the Iceshelfas a result of poor enrollment in india and it is

entrenched truth that over 85% of removals are gone before by minor DFU which get contaminated. The mortality chance is most noteworthy in patients with unending osteomyelitis also, in those with intense necrotizing mild tissue diseases.

PATHOPHYSIOLOGY

Patients suffering from long standing diabetes are,for the most part are vulnerable to foot infections predominantly because of Immunopathy (diminished neutrophil function),Vasculopathy (vascular insufficiency) and Neuropathy. 50 to 70 percent of diabetic patients with foot infection is found to haveperipheral neuropathy which plays a significant role in the development ofcomplications. Patients with diabetes lose the defensive sensations for temperature and agony, debilitating consciousness of injury, for example, development of blisters, minor abrasions, or penetrating injury with foreign body. Exposed feet walking is exceptionally more common in Indians especially those of village areas and with insensate foot diabetic patients do not come to know difference of wounds made by little pebbles,thorn,Splinters,nails etc.Motor neuropathy canresult in foot distortions (e.g., Hammer and paw toe and so forth.) that add to build neighborhood weight from footwear, prompting corn or callus production with subcutaneoustissue making skin ulceration much more probable.

Once the skin is broken (regularly on the plantar surface), the deeper tissues are presented to colonization by pathogenic microbes . The subsequent tissue damage due to the injury in patients with disease may start externally, in conjunction with delay in treatment and impeded body resistance mechanisms, it can spread to the adjacent subcutaneous tissues and to significantly involving more underlyingstructures(deep Plantar spaces) .Although most diabetic foot contaminations start with a ulcer, cellulitis and necrotizing fasciitis can develop without a ulcer or horrendous damage. Most diabetic foot diseases happen in the setting of good dorsalispedispulse ; this finding shows that the essential issue in diabetic foot contaminations is micro vascular compromise. On the off chance that long standing chronic osteomyelitis is left untreated for quite a long time, it might lead to the development of amyloidosis or squamous cell carcinoma at the site of skin drainage.

MICROBIAL CHARACTERISTICS

The microbiologic characteristics of diabetic ulcer foot infections vary according to the type of tissue getting infected. Infections such as cellulitis in diabetic patients is are infact caused by organisms which commonly infect healthy hosts like staphylococcus aureus. Group B streptococcal cellulitis is infrequent in healthy hosts but not in patients with diabetes, Group B streptococci may cause urinary tract infections

and catheter-associated bacteriuria In patients with diabetes in addition to cellulitis, superficial or deep soft-tissue infections, and chronic osteomyelitis. Bacteremia complicatesthese kind of infections.Moreover, as formerlycited, deep soft-tissue infections in patients with diabetes can be accompanied with gas-producing, Gram-negative bacilli. Clinically, these infections appear as necrotizing fasciitis, compartment syndrome, or myositis. Gas gangrene is not so common in diabetic patients. An individual with diabetes generallydevelop acute osteomyelitis as a consequence of foot trauma.The spectrum of infecting organisms is the same as that in an individual without diabetes who is suffering from acute osteomyelitis. In chronic osteomyelitis, however, the micro organismslike group A and group B streptococci, aerobic gram-negative bacilli, and Bacteroidesfragilis were predominantly present. B.fragilis, Escherichia coli ,Proteus mirabilis and Klebsiella pneumoniae were theother organismslinked in chronic osteomyelitis in patients with diabetes. Pseudomonas aeruginosa is in generalis not a micro organismin chronic osteomyelitis in those individuals. Although P aeruginosa is commonly cultured from samples acquired from a draining sinus tract or deep penetrating ulcers in diabeticpatients, these organisms are superficial colonizers and are usually not the cause of the bone infection. Because Pseudomonas pathogens are water-borne, superficial ulcers may be infected by bacteria in dressings ,wet socks or walking bare feet in

spiritual places (a communal practice embraced by Indians washing their feet in common water channel while going inside Temple ,church or Mosque) or common swimming pool or bathing places. Anerobic micro-organisms causes a combined deep-skin and or softtissue infectionrepresents fetid foot. Anaerobics are frequently part of mixed infections in diabeticpatients with foot ischemia or gangrene. The most common pathogens in acute, previously untreated, superficial infected foot wounds in patients with diabetic foot are aerobic gram-positive bacteria, predominantlyBetahemolytic streptococci (group A, B, and others)and Staphylococcus aureus.Those infections in diabetic patients who have recently received antibiotics or who have deep limbthreatening infection or chronic wounds are caused usually by a combination of aerobic gram-positive, aerobic gram-negative (e.g., Proteus species ,Escherichia coli , Klebsiella species), and anaerobic organisms (e.g., Clostridium species Bacteroides species, Peptococcus and Peptostreptococcus species).Methicillin-resistant S. aureus (MRSA) is a more common micro organism in patients who have been earlier hospitalized or who have in recent timesreceived anantibiotic therapy. MRSA infection can also occur in the nonexistence of risk factors because of the growingfrequency of MRSA in the community.

ESTABLISHING SEVERITY OFINFECTION

The severity of the disease decides the selection ofsuitable antibiotic regimen and the route in which they are administered. It additionally is the essential factor in deciding the requirement for hospitalization and the planning of any surgery warranted.

CLINICAL CLASSIFICATION OFDIABETIC FOOT INFECTION

IDSA (**Infectious Diseases Society of America**) Diabetic Foot

Infection Classification:

- **Uninfected:** lacking purulence or signs of inflammation
- **Mild:** infection limited to superficial tissue, cellulites < 2 cmaround ulcer, no systemic signs
- **Moderate:** Systemically well & metabolically stable, more thanone of -Cellulites > 2 cm from ulcer, involvement of deep tissue,gangrene, abscess, muscle, tendon, joint or bone involvement
- **Severe:**infection of foot with metabolic instability and/or systemic toxicity
- Hypotension ,Tachycardia
- Vomiting , Confusion
- Fever or chills
- Leukocytosis
- Severe hyperglycemia, azotemia or DKA

ESTABLISHING THE EXTENT OFINFECTION

Diabetic Foot infections are like Iceberg, Most of the time only smallpart is visible.Early recognition of the area of involved tissue can facilitate appropriate management and prevent progression of the infection. The wound should be cleansed and debrided carefully to remove foreign bodies or necrotic material and should be probed with a sterilemetal instrument to identify any sinus tracts, abscesses, orinvolvement of bones or joints.Osteomyelitis is a common and serious complication of diabetic footinfection that poses a diagnostic challenge. A delay in diagnosisincreases the risk of amputation.Risk factors associated with osteomyelitis are summarized in Visible bone and palpable bone by probing are suggestiveof underlying osteomyelitis in patients with a diabetic foot infection.Laboratory studies, such as erythrocytes sedimentation rate (ESR)white and blood cell count have limited sensitivity pattern in the diagnosis of osteomyelitis. Osteomyelitis is unlikely with normal ESR values; however, an ESR of more than 70 mm per hour supports a clinicalsuspicion of osteomyelitis.Definitive diagnosis requirespercutaneous or open bone biopsy. Bone biopsy is suggested if imaging studies are doubtful in the diagnosis of osteomyelitis.

CLINICAL EVALUATION

Treatment

Effective management of diabetic foot infection requires

- Appropriate antibiotic therapy,
- Surgical drainage, debridement and resection of dead tissue,
- Appropriate wound care and
- Correction of metabolic abnormalities.

ANTIBIOTIC THERAPY

The choice of antibiotic therapy for diabetic foot infection involvesdecisions about

- Selection of empiric and definitive antibiotic drugs
- Route in which they are administered
- Period of treatment.
- The choice of empiric antibiotic regimeought to include drugs which are activeagainst *Staphylococcus aureus*, including methicillin-resistant *Staphylococcus aureus* if necessary, and also streptococcal organisms.
- In patients withsevere infection, chronic infection, or infection that fails to respondto recent antibiotic therapy coverage for aerobic gram-negative bacterias is also required.

- Antibiotic therapy iscommonly required for anaerobic organisms causingwounds which are foul-smelling necroticor gangrenous.
- Upon the foundationof clinical response of the treatment and culture or susceptibility testing ,the choice of initial empirical antibiotic treatment should be altered.
- In patients with polymicrobial infection, coverage for virulent organisms, such as *S. aureus* and streptococci, is strongly recommended.
- Treatment for less virulent organisms, such as Coagulase-negativestaphylococci, may not be required.
- Parenteral antibiotics are specified for diabetic patients who have systemic illness, or if severe infection persists, patients who are unable to tolerate oralagents, or have infection caused by organisms that are notin the spectrum of coverage to oral agents.
- Utilizing oral anti microbial agents for mild to moderate infection and changing right on time from parenteral to oral anti-microbials with suitable rangeof spectrum and great bioavailability and tolerance are firmly supported.
- Although topical antibiotics can be active for the management ofmildly infected ulcers, they should not be regularly used.

- When all the signs and symptoms of infection has been resolved ,termination of antibiotics should be considered , even if the woundhas not entirelycured.
- Cost effectiveness is an important factor in the choiceof selecting antibiotic treatment.Preliminary empiric antibiotic therapy should be based on the virulence ofthe infecting organisms , any history of recent antibiotic treatment, previous infectionwith resistant organisms, latest culture results, recent Gram stainfindings, and patient factors (e.g., drug allergyetc).

A Gram-stained smearof ansuitable wound specimen may help tomonitor therapy. Theglobal sensitivity of a Gram-stained smear for identifying pathogensthat grow on culture mediais 70 percent. The empiric antibiotic therapyfor diabetic foot infection should always include an effective drug activeagainst *S. aureus*, including MRSA if necessary, andstreptococci.5,26,27,28 .The patient should be reassessed 24 to 72 hours after initiating empiricantibiotic treatment to assess the response and to alter the antibioticregimen, if indicated by initial culture results. Several antibiotics havebeen shown to be active, but no single regimen has shownsuperiority.Antibiotic treatment should not be used for footulcers without signs of infection since those regime do not enhance woundhealing or aid in the prevention of infection.

Clinical catastrophedespite of appropriate antibiotic treatment might be because ofpatient nonadherence, superinfection ,undiagnosed deep abscess antibiotic resistance or osteomyelitis or severe tissue ischemia.

TABLE 6 : ANTIMICROBIAL REGIMEN

CATEGORY	ANTIMICROBIAL REGIMEN
NON-LIMB THREATENING	Cephalexin 500mg p.o.q6h Clindamycin 300 mg p.o.8h Amoxicillin Clavulanate (875/125 mg)1q12h Dicloxacillin 500mg q6h Levofloxacin 500-750mg qd
LIMB THREATENING	Ceftriaxone 1g IV daily plus Clindamycin 450-600 mg ivq8h Ciprofloxacin 400mg iv q 12h plus Clindamycin 450-600 mg ivq8h Ampicillin/sulbactam3g iv 6h Ticarcillin/clavulanate3g iv q4-6h Piperacillin/tazobactam 3.375 g q4h or 4.5g iv q6h Flouroquinolone iv plus metronidazole 500mg iv q8h
LIFE THREATENING	Imipenemcilastatin 500mg iv q6h Piperacillin/tazobactam 4.5g iv q6h plus Gentamicin 1.5mg/kg iv8h Vancomycin 1g ivq12h plus Gentamicin plus metronidazole

SURGICAL TREATMENT

Surgery is the cornerstone of treatment for deep seated diabetic footinfection. Timely and aggressive surgical debridement or limitedresection or amputation may reduce the need for more extensiveamputation.34 Procedures range from simple incision and drainage toextensive multiple surgical debridement and amputation. Indicationsfor emergent surgery are severe infection in an ischemic limb,necrotizing fasciitis, gas gangrene, and an infection associated withcompartment syndrome. In diabetic patients with osteomyelitis surgical removal of affected bone has been the customary treatment.In two thirds of patients with osteomyelitis , successful treatment with a long course of antibioticsalone has been achieved in the outcome of diabetic foot infections treated conservatively as evident by aretrospective cohort study with long-term follow-up. As infectionis controlled and the wound starts to granulate, delayed primaryclosure may be successful. The wound may also be treated surgicallywith a flap or graft, left to heal by secondary intention, or managedwith negative pressure dressings,NPWT or VAC(Vacuum AssistedClosure). The patient should be managed by a vascular surgeon if ischemic changes appear in the infected limb. Although noncritical ischemiacan usually be treated without a vascular procedure, promptrevascularization within short period of onset of the

infection is required forsuccessful treatment of an infected foot with critical limb ischemia.

SURGICAL DECOMPRESSION

it comprises of 3 types

- forefoot decompression
- plantar space decompression
- foot and leg decompression

FOREFOOT DECOMPRESSION

Web space infection,central plantar space infection are the indications. Incision should be placed deep into plantar space cutting plantar aponeurosis.

PLATAR SPACE DECOMPRESSION

Main indication is plantar space infection.Characteristic factor of this abcess is disappearance of longitudinal arch and skin crease.The area of longitudinal arch may bulge, sole is edematous,incision is made from little toe to heel over the medial aspect.

FOOT AND LEG DECOMPRESSION

Vertical incision for leg and horizontal for foot abscess , cellulitis are done.

AVERAGE HEALING TIME

Forefoot decompression - 11-38 days

Plantar decompression - 12-40 days

Foot and leg decompression - 12- 60 days

ROLE OF AMPUTATION

Factors which are considered in the decision of amputation are

1. Age
2. Nephropathy
3. Major vessel disease
4. Gross neuropathy
5. Presence of gangrene
6. Involvement of bone
7. Uncontrolled diabetic ketoacidosis
8. Septicemia

TYPES OF AMPUTATION

1. Toe amputation
2. Great toe amputation
3. Other toes amputation
4. Ray amputation
5. Trans metatarsal amputation
6. Below knee amputation

VASCULAR MANAGEMENT

1. Role of pentoxyphylline
2. Antiplatelet drugs
3. Surgery – Endarterectomy / Bypass procedure

WOUND MANAGEMENT

Dressing of the wound has to in order be adequate and to done in a proper manner to allow for careful examination forindication of healing and in prompt identifying any new necrotic tissue.Necrotic or unhealthy tissue should be debrided, surgicallyor with topical debriding agents. Eliminating pressure from the footwound (i.e.Off-loading) is crucial for healing which can be achieved through total contact casting, detachable castwalkers, and various ambulatory braces,modified half-shoes,splints and sandals. Edema of the legs can delay wound healing which inturn can be managed with leg elevation, compression stockings, or a pneumaticpedal compression device whichenhances the healing process.resolution of infectionis evident fromestablishment of granulationtissue, lack of necrotic tissue, and concluding of the wound. Ifosteomyelitis is present, signs of healing include a drop in ESR &CRP and loss of increased uptake on nuclear scan.

ADJUVANT THERAPY IN WOUND HEALING

1. Cultured human dermis and cultivated equivalents
2. Hyperbaric oxygen therapy
3. Ketanserin
4. Growth factors
5. Granulocyte – colony stimulating factor
6. Electrical stimulation
7. Sulodexide
8. Hyaff
9. Low level laser therapy

RECOMMENDATIONS

- The feet should be examined atleast annually in patients with type 2 diabetes and in those with type 1 diabetes for more than 50 years
- A detailed neurological examination and assessment for peripheral vascular disease should be performed
- We recommend using the quantitative foot assessment for neurological symptoms and signs described above,including the 5.07 u monofilament test
- Patients should be considered particularly high risk for future plantar ulceration if they have any
- A previous history of foot ulceration or amputation
- Neuropathic foot deformities ,especially with overlying bunions oe calluses.

INDICATIONS OF HOSPITALIZATION

A clinician should remember certain indications of Hospitalization in Diabetic Foot Infections like-

- Serious Infection like Necrotising Fasciitis/Gas gangrene
- Patients who require Parenteral Therapy or fluid resuscitation
- Patients who need Surgical intervention
- To regulate metabolic instabilities e.g. Diabetic Ketoacidosis
- Patient who is unable or not willing to perform proper wound care
- Patient who can or will not be able to offload the wound

METABOLIC STABILITY

Adequate glycemic control aids in the eradication of infection and supportwound healing. At initial presentation the blood glucose and hbA1Clevels measured in all diabetic patients and then at fixed intervals.Frequent home blood glucose monitoring is strongly encouraged.Other than Blood sugar control correction of fluid and electrolyteimbalances, acidosis, and azotemia is essential.Patient's nutritionshould be taken care of particularly high protein diet(If no renalproblem).

PROGNOSIS

The prognosis for cases of cellulitis, skin and/or soft-tissue infections,and acute osteomyelitis depends on the adequacy of antimicrobialtherapy and surgical debridement. For cases of chronic osteomyelitis,the prognosis is directly related to the vascular supply in the affectedlimb and the adequacy of surgical debridement along with adequateoff loading.

PREVENTION

Prevention of diabetic foot ulcers begins with identifying patients at risk. All patients with diabetes should have an annual foot examinationthat includes assessment for anatomic deformities, skin breaks, naildisorders, loss of protection sensation, diminished arterial supply,and inappropriate footwear. Patients at higher risk of foot ulcerationshould have examinations more often.42 Educating patients andcaretakers about proper foot care and periodic self-foot examinations are effective interventions to prevent ulceration. Other effective clinicalinterventions include optimizing glycemic control, smoking cessation, debridement of calluses and certain types of prophylactic footsurgery.

PROPHLACTIC FOOT CARE

It is important that prophylactic advice on foot care can be given to any patient whose feet are at high risk. The recommendations foe prophylactic foot care are :

AVOID

1. Smoking.
2. Walking bare foot.
3. The use of heating pads or hot water bottles.
4. Stepping into a bath without checking the temperature.

   The feet should be

   - Washed daily in tepid water
   - Mild soap should be used and the feet should be dried by gentle patting
   - A moisturizing cream or lotion should then be applied.

Toe nails :

- Trimmed to the shape of the toe
- Filed to remove sharp edges

Shoes :

- The patients shoes should be snug ,not tight
- Patients who have misshapen feet or have had a previous foot ulcer may benefit from the use of special customized shoes.
- A prospective study found that shoe variables other than the recommendation for customized shoes (e.g,.style,width,length,or type of shoe) had no preventive effect.
- The use of customized shoes,however,reduced the development of new foot ulcers from 59 to 27 percent over one year of follow up in a study report.

Socks :

- Cotton
- Loose fitting
- Should be changed every day.

Inspection of feet :

The feet should be inspected daily, looking between and underneath the toes and at pressure areas for skin breaks, blisters, swelling, or redness. The patient may need to use a mirror or if vision is impaired, have some one else perform the examination.

Examination of foot by medical personnel :

A particularly effective strategy is to make specific recommendations to the patient in the form of a " contract". And to advise the patient to request that his or her feet has to be examined at every visit to the doctor.

IDEAL DIABETIC FOOT TEAM

Diabetic ulcer foot management requires a team work focusing on preventing complications and salvaging the foot. The team includes :

- General surgeon
- Diabetician / dietician
- Podiatrist
- General physician
- Microbiologist
- Pharmacist
- Nurse
- Orthopediacian
- Vascular surgeon
- Physiotherapist
- Laboratory sprecialist (foot pressures)

MATERIALS AND METHODS

Study design

This prevalence study is to determine the type of bacterial microorganisms causing chronic diabetic ulcer foot in Diabetic patients attending Coimbatore Government Medical College Hospital, Coimbatore, Tamil Nadu. This study also aims at the antimicrobial susceptibility pattern of the bacteria isolated from the ulcer wounds.

Source of sample and questionnaire

The duration of the study is for two months during the period from January 2017 to January 2018. A total of 50 patients suffered from diabetic foot ulcer were registered in this study. Consent were obtained from the patients with diabetes proirly. Information regarding patients demographic and clinical features such as Name, Age, Sex, Type of Diabetics, Duration since first diagnosis, Duration of stay in the hospital, Glycemic control during treatment, Size of the ulcer, presence of family history, awareness aboutthe complications, personal history(habit of smoking /alcoholic), History of hypertension in the past, peripheral neuropathy, trauma and other peripheral vascular diseases. Clinical conclusions are formulated for each of the patient. Clinical valuation of signs and symptoms (pyrexia swelling ,tissue necrosis, exudates, odour,

surrounding cellulitis, crepitation) is made. Ulcer size is established by multiplying the longest and widest diameter.

Sample Processing:

Using 2 sterile swabs,samples were collected deep from the base of the diabeticulcer foot. Of the twoswabs one was used for culture andthe other forgram staining.The specimens collected were cultured onto culture media namely blood agar, chocolate agar, Mac Conkey's agar and Thioglycollate medium .The samples were incubated at 37◦ C overnight .The plates were examined for growth on thethe next day. The organisms were recognizedbased on the direct gram staining, morphological characteristics of the colonyand theirbiochemical reactions.

Inclusion criteria

Type 2 diabetes mellitus Patients with chronic ulcers are included.

- ✓ Age : 30- 80 yrs

Exclusion criteria

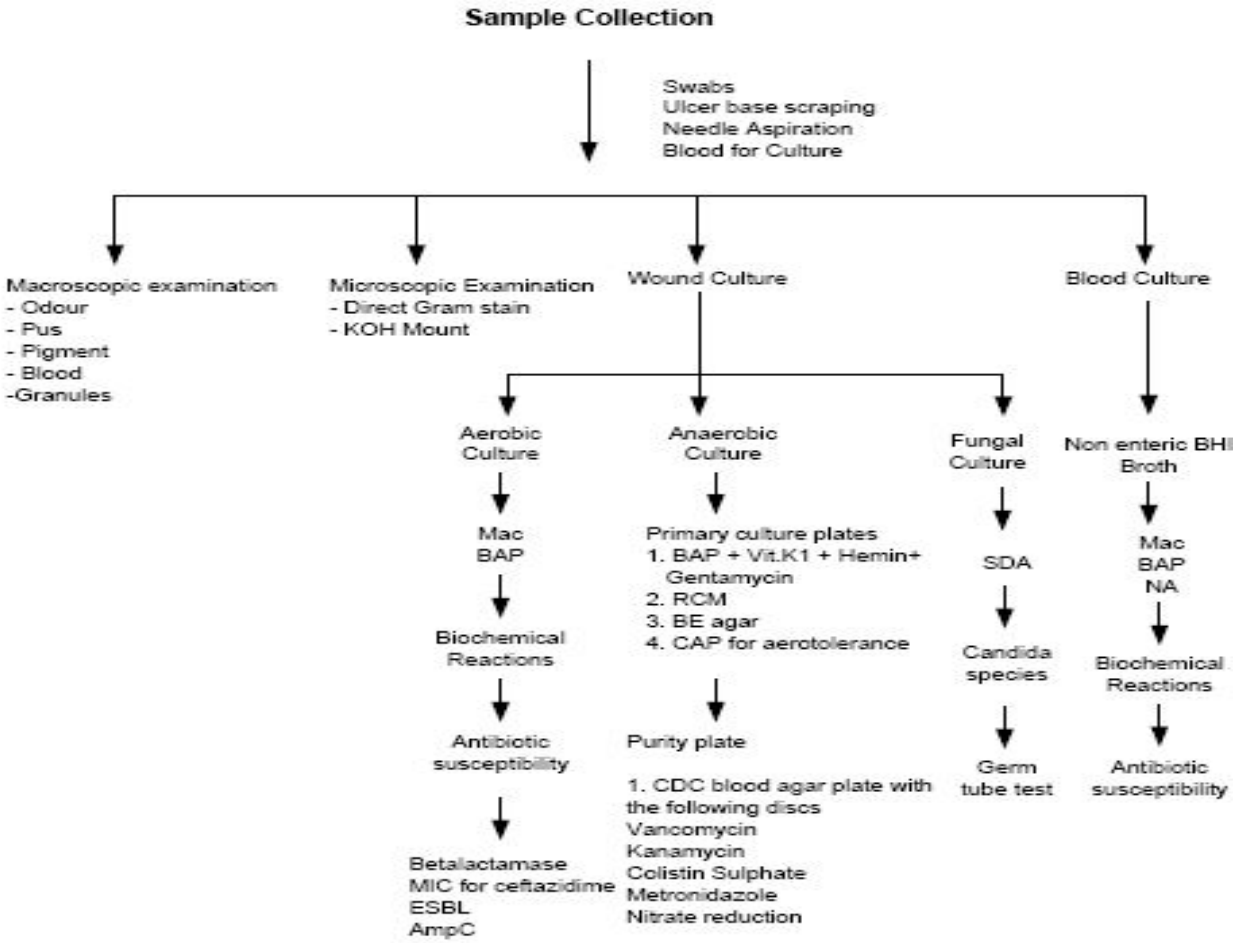
- ❖ Patients who has been started with antibiotic treatment for diabetic foot ulcer.
- ❖ Age less than 30 years and more than 80 years
- ❖ Pregnant women
- ❖ Ulcer involving vasculopathy and neuropathy

- ❖ Ulcer with osteomyelitic changes
- ❖ Ulcer over long standing scar

Microbiological methods used

Wound specimens such as pus, and wound exudates are collected from the patient. For ulcer, before sampling the wound was debrided with a sterile scalpel and rinsed with sterile normal saline. Specimens are obtained by deep swabs or aspirating the pus from abscesses. When obtaining a specimen for culture and sensitivity testing, it is a key to collect material that is not contaminated withcolonizing flora, but contains true pathogens. Since false-negative cultures can occur due to previous antibiotic therapy , it is best if specimens can be obtained before such therapy is begun. The specimen is positioned in an appropriate sterile transport container and swiftly sent to the microbiology laboratory for bacterial isolation. Each specimen is streak inoculated and incubated aerobically at 37◦c overnight. The organisms are isolated in pure culture on the solid media.

TABLE 7 : SAMPLE COLLECTION PROCESS



Antimicrobial susceptibility testing

The antimicrobial susceptibility of micro-organisms is done by Kirby-Bauer disc diffusion method with the necessary discs as recommended by Central Laboratory Standard Institute (CLSI) . 4to 5 similar looking colonies are picked and inoculated into peptone water. The density of the broth is adjusted to by comparing its turbidity with that of 0.5 McFarland opacity standard tube. It is inoculated on the Muller Hintoin Agar medium with the antibiotic discs and left overnight to obtain semi confluent growth. The zone of inhibition and sensitivity are measured after 18hours and noted down. Gram negative bacteria are also tested for extended spectrum Beta lactamases (ESBL) production by screening test as recommended by Central Laboratory Standard Institute (CLSI). ESBL production can be tested by screening methods and confirmatory texts. In this study screening for ESBL production is done by using cefpodoxime (mg) discs along with other 3rd generation cephalosporin discs . And the Staphylococcal species are tested for Methicillin Resistance (MRSA and MR-CONS) by using cefoxitin disc along with the recommended drugs for *Staphylococcus*.

OBSERVATION AND RESULTS

Distribution according to age and sex

A total of 50 patients who have chronic diabetic foot ulcer are included in this study. Out of 50 patients, 37 are male patients (75%) and 13 are female patients (25%). The mean average age of the patients is 59.2. Estimated ratio of male and female is 3:1. The age of patient ranges between 30 and 80. Here maximum number of affected patients lies between of age group 51-60 of both male and female are alike.

It is followed by the age group 61-70(22%) with males 73% and females 27%, 71-80(18%) with male 83% and female 18%, <40(12%) with males 83% and females 12%. The least affected group is >81(2%) in which only males are affected. It is summarized in table 1 and 2. Of the 52 diabetic patients, 31(60 %) patients had random blood sugar more than 200 mg/dL.

Microbes isolated from culture

Out of 52 isolates, 50 are found to have growth. Aerobic Gram-negative bacteria were the most commonly isolated micro organisms and accounted for 94% of all isolates and gram positive which accounts for 6%.

The most common individual isolates was*Escherichia coli* *Klebsiella pneumoniae* (47%). It is followed by *Pseudomonas aeruginosa* (19%), *Proteus mirabilis* (15%), *Citrobacter* sp. (6%), (6%), *Acinetobacter* sp. (2%), *Staphylococcus aureus* (3%). It is summarized in table 19.

Antimicrobial susceptibility

Klebsiella pneumoniae

*Klebsiellapneumoniae*is frequently sensitive to antimicrobials such as Imipenem (90%) cefoperazonesulbactum (79%), Piperacillintazobactum (63%), chloramphenicol (60%), and sensitivity to Cefepime was only about 29%. Klebsiella pneumonia was resisitant to most of the Cepalosporins, Fluoroquinolones and Co trimoxazole; the incidence of resistance is as follows; Cefuroxime (92%), Ampicillin sulbactum (90%), Amoxicillin clavulanic acid (90%), Cefpodoxime (90%). It is summarized in table 14.

Proteus mirabilis

All the proteus mirabilis isolates are more sensitive to Imipenem and Cefoperazone sulbactum. It is followed by Piperacillintazobactum (93%). Proteus is resistant to Levofloxacin (87%) and Cotrimoxazole (87%). It is summarized in table 15.

Pseudomonas aeruginosa

Pseudomonas aeruginosashows highest sensitivity to antimicrobials like Imipenem (89%), followed by Piperacillintazobactum (79%), Cefoperazone-sulbactum (68%), Tobaramycin (60%) and resistance to antimicrobials such as Levofloxacin (89%), Ofloxacin (84%), Gentamicin (74%). It is summarized in table16.

Escherichia coli

Escherichia coli is more sensitive to antimicrobials like Imipenem, Cefoperazone sulbactum. They all are found to be resistant to antimicrobials like Cotrimaxazole and Cephalosporins. It is summarized in table 17.

Staphylococcus aureus

Highest sensitivity is found to Vancomycin. The most resistant antibiotics are Cotrimoxazole, Cipro/levo/of, Gentamicin, Amoxicillin clavulanic acid. *Staphylococcus* which are isolated are resistant to Penicillin (67%). They are also found to have Methicillin resistance (resistant to Cefoxitin 67%). It is summarized in table 18.

A very few *Acinetobacter*species are also isolated. They are found to be resistant to all the antimicrobial discs tested.

TABLE 8 :AGEWISE DISTRIBUTION

Age groups	Male		Female		Total (N=50)	
	N	%	N	%	N	%
<40	5	83	1	17	6	12
41 – 50	5	56	3	44	8	16
51 – 60	12	75	4	25	16	32
61 – 70	8	73	3	27	11	22
71 – 80	6	83	2	17	8	16
>81	1	100	0	0	1	2

CHART 1 : AGE WISE DISTRIBUTION

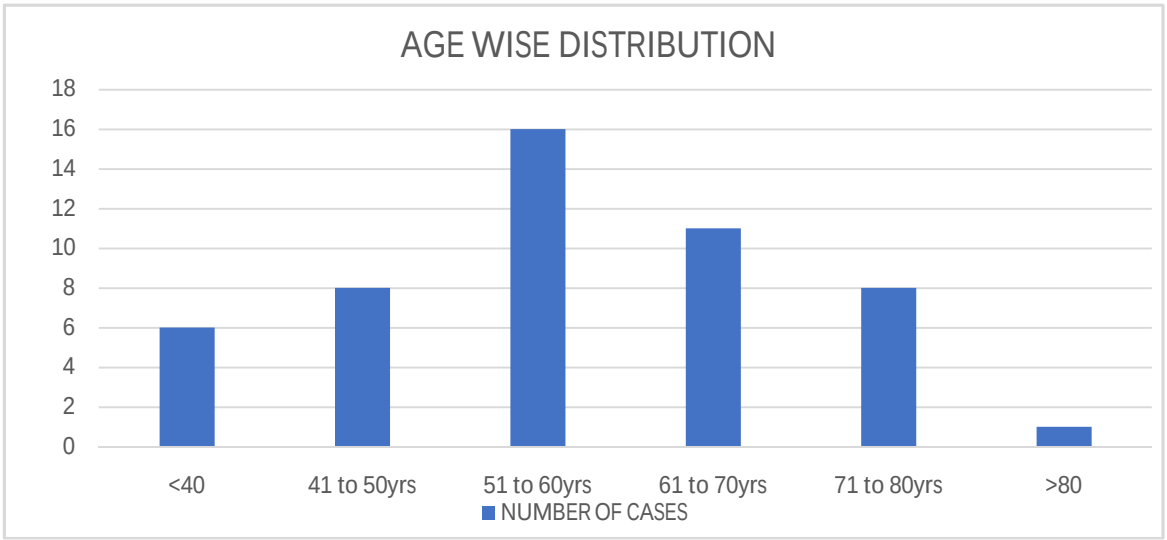


TABLE 9 :SEX DISTRIBUTION

SEX	NUMBER OF CASES	PERCENTAGE
Male	37	**74.00%**
Female	13	**26.00%**
Total	50	100.00%

CHART 2 : SEX DISTRIBUTION

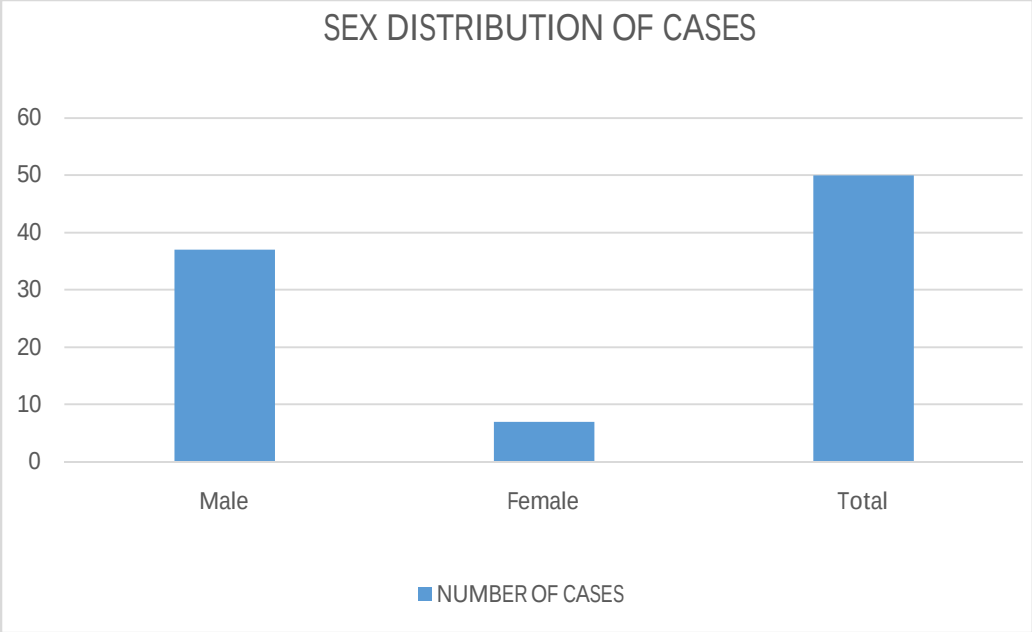


TABLE 10 :DISTRIBUTION BY DISEASE DURATION

DISTRIBUTIONBYDISEASEDURATION						
DURATIONI NYEARS					TOTAL	PERCENTAGE
	NO.	PERCENTAGE	NO.	PERCENTAGE		
≤5	1	41	0	0	1	1
6TO10	2	35	1	4	3	3
11 T0 15	7	20	2	12	9	17
16TO 20	11	2	4	37	15	36
≥20	16	1	6	47	22	44
Total	37	100	13	100	50	100

CHART 3 : DURATION OF DISEASE Vs POLYMICROBIAL ISOLATES

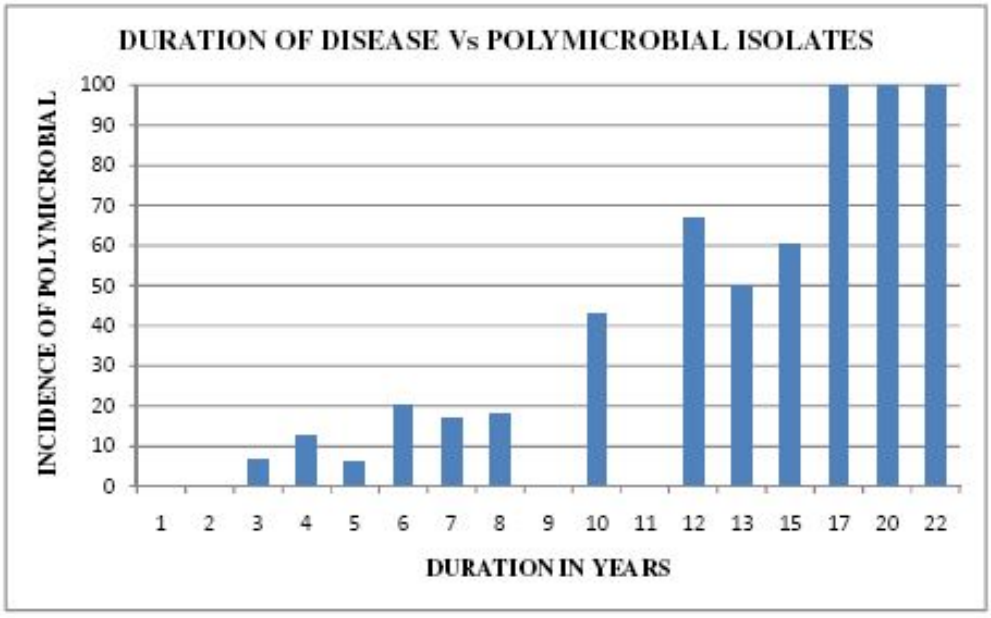


CHART 4 : DURATION OF DISEASE Vs MDRO

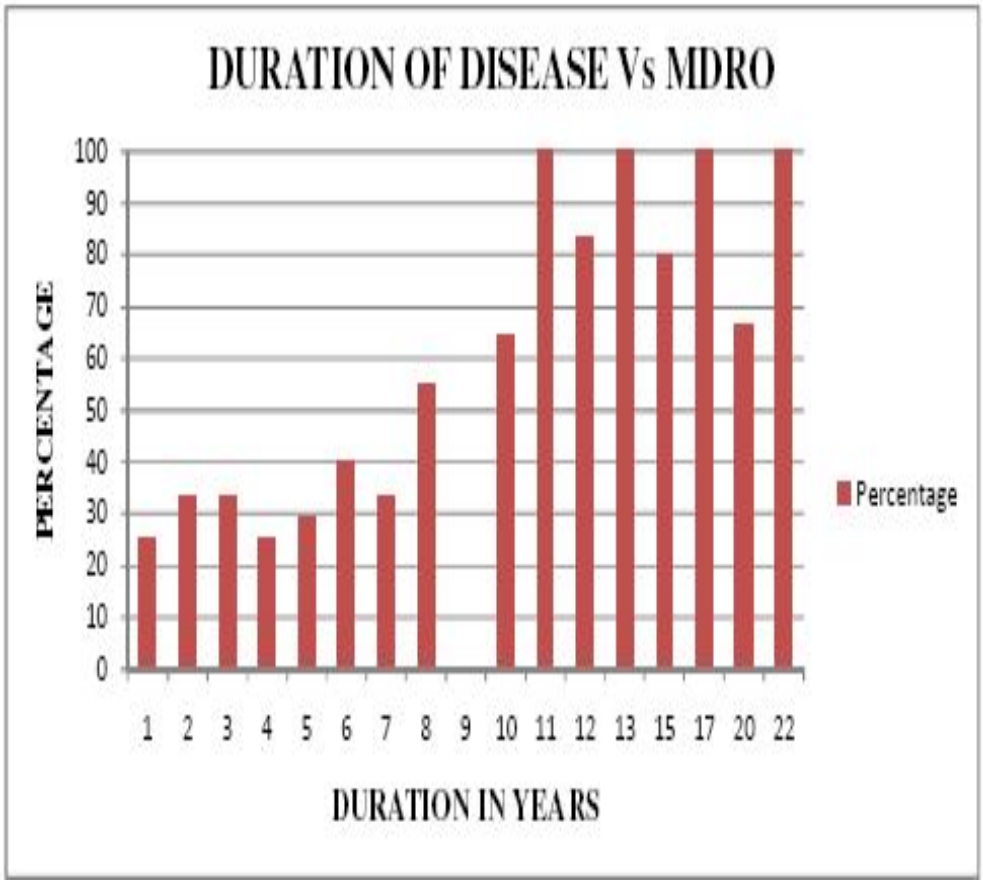


TABLE 11 :ASSOCIATED COMPLICATIONS

ASSOCIATEDCOMPLICATIONS		
DISEASE	**NUMBEROFPATIENTS**	**PERCENTAGE**
NEUROPATHY	39	78
NEPHROPATHY	18	36
PERIPHERALVASCULARDISEASE	38	76
RETINOPATHY	18	36
HYPERTENSION	33	66

CHART 5 :ASSOCIATED COMPLICATIONS

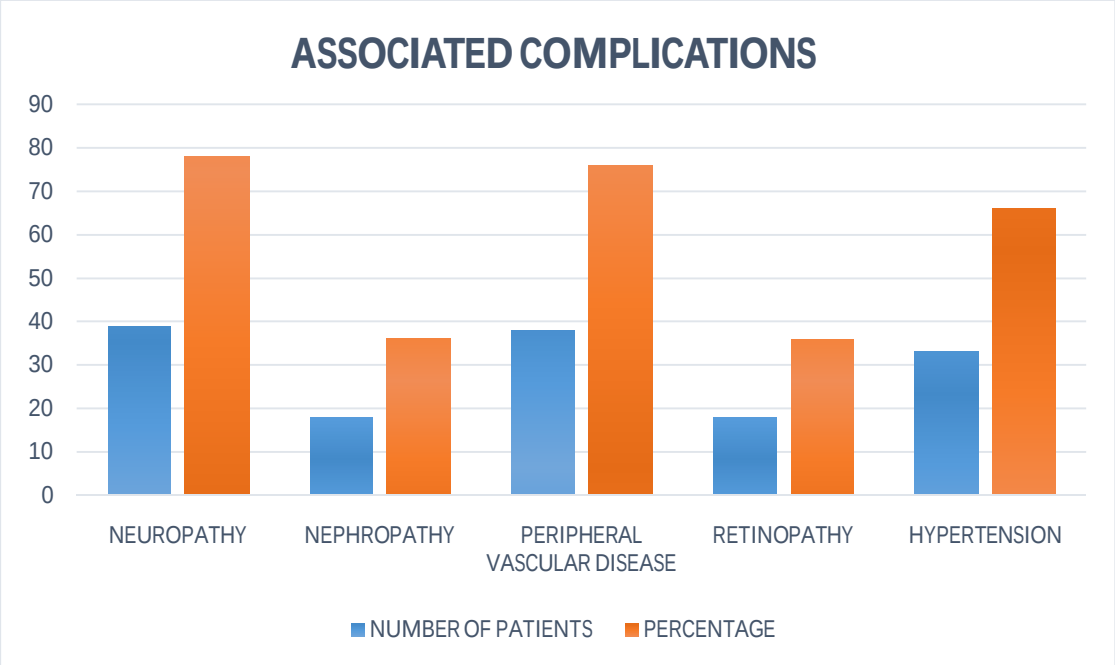


TABLE 12 : CORRELATION BETWEEN GLYCEMIC CONTROL & AMPUTATION

Glycemic control	Total	MDRO		lymicrobialinfection		Amputation	
		No.	Percentage	No.	Percentage	No.	Percentage
FBS≥110	36	14	48	10	20	1436	48
PPBS≥160	14	20	40	14	28	36	72

TABLE 13 : CLINICAL OUTCOME

	CONSERVATIVE	AMPUTATION
TOTALPATIENTS	36	14
PERCENTAGE	72	28

CHART 6 : CLINICAL OUTCOME

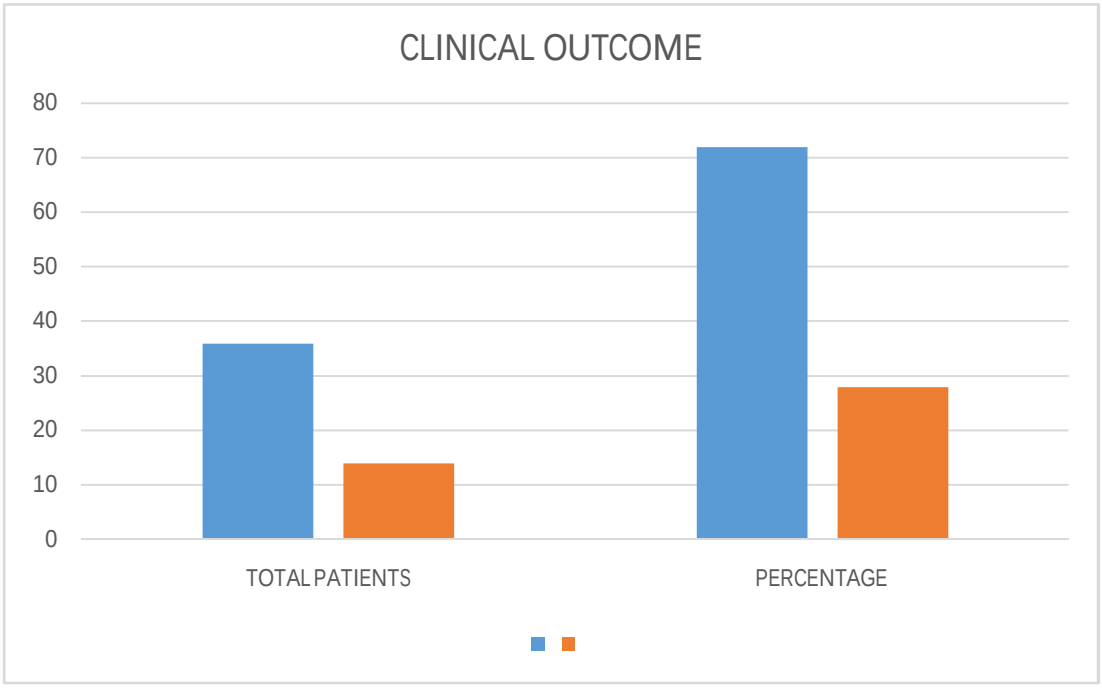


TABLE 14 :CORRELATION BETWEEN MDRO & AMPUTATION

	AMPUTATION	MDRO	POLYMICROBIALGROWTH
NO.OFPATIENTS	14	34	24
PERCENTAGE	28	68	48

CHART 7 : CORRELATION BETWEEN MDRO & AMPUTATION

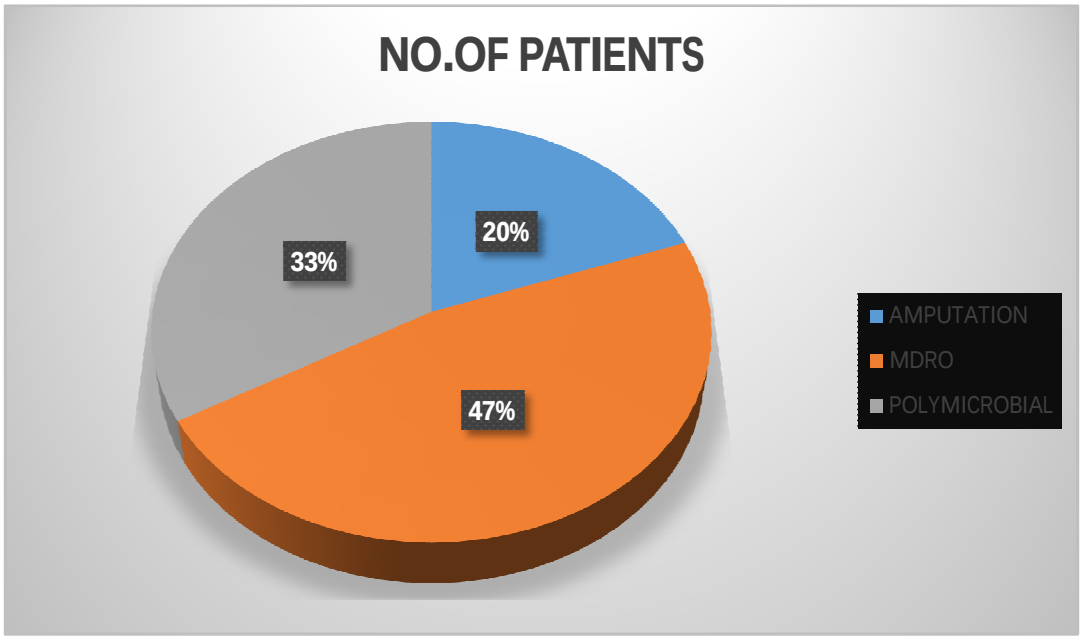


TABLE 15 :ANTIMICROBIAL SUSCEPTIBILITY OF *KLEBSIELLAPNEUMONIAE*

Name	Sensitive	Resistant
Gentamicin	13	37
Amikacin	20	30
Cefuroxime	4	46
Ceftriaxone	13	37
Cefepime	19	31
Cefpodoxime	5	45
Cefoperazonesulbactum	39	11
Ciprofloxacin	8	42
Levofloxacin	11	39
Cotrimoxazole	13	37
Chloramphenicol	30	20
Imipenem	45	5
Ampicillinsulbactum	5	45
Amoxicillinclavulanicacid	5	45
Piperacillin Tazobactam	31	19

CHART 8 :ANTIMICROBIAL SUSCEPTIBILITY OF

KLEBSIELLA PNEUMONIAE

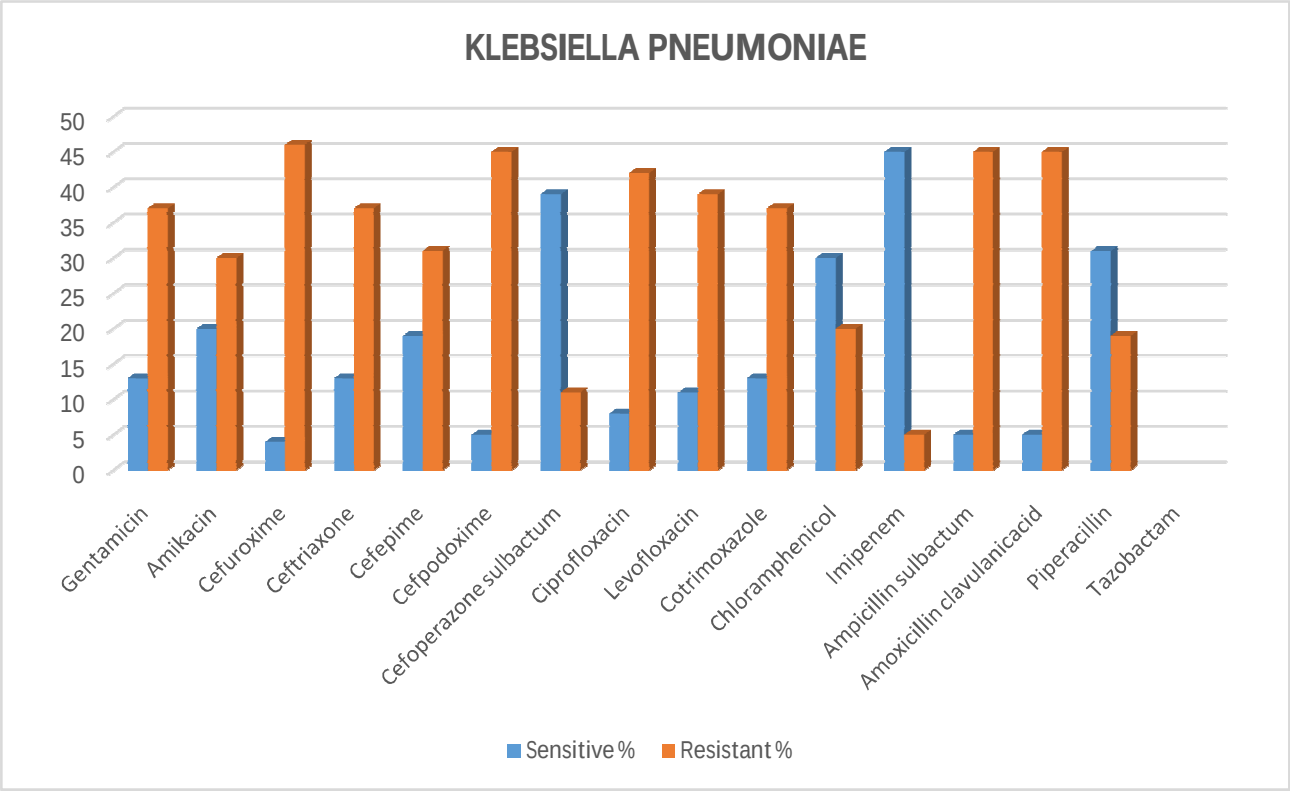


TABLE 16 :ANTIMICROBIAL SUSCEPTIBILITY OF *PROTEUS MIRABILIS*

Name	Sensitive	Resistant
Gentamicin	20	30
Amikacin	20	30
Cefuroxime	17	33
Ceftriaxone	20	30
Cefepime	30	20
Cefpodoxime	10	40
Cefoperazone sulbactum	50	0
Ciprofloxacin	10	40
Levofloxacin	7	43
Cotrimoxazole	7	43
Chloramphenicol	26	24
Imipenem	50	0
Ampicillin sulbactum	17	33
Amoxicillin clavulanicacid	17	33
Piperacillin Tazobactam	47	3

CHART 9 :ANTIMICROBIAL SUSCEPTIBILITY OF *PROTEUS MIRABILIS*

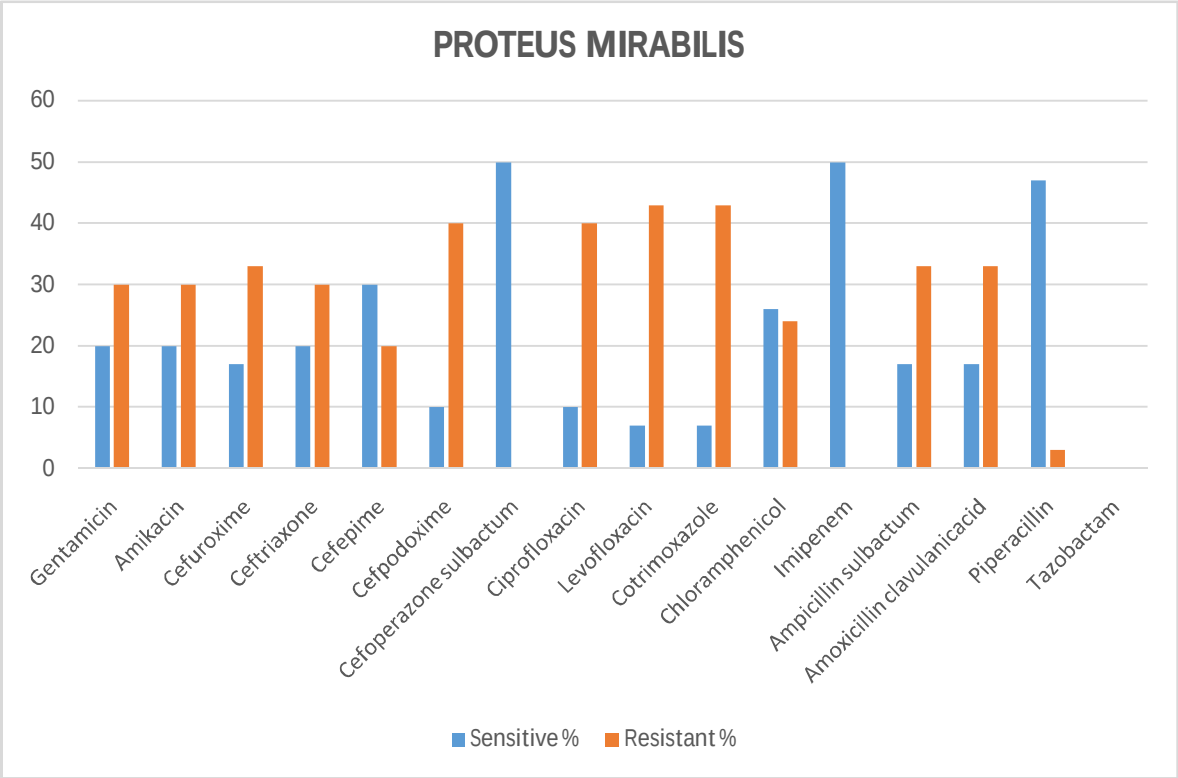


TABLE 17 :ANTIMICROBIAL SUSCEPTIBILITY OF *PSEUDOMONAS AERUGINOSA*

Name	Sensitive	Resistant
Gentamicin	13	37
Amikacin	26	24
Cefuroxime	8	42
Ceftriaxone	12	38
Cefepime	16	34
Cefpodoxime	10	40
Cefoperazonesulbactum	39	11
Ciprofloxacin	29	22
Levofloxacin	7	43
Imipenem	41	9
Tobramycin	30	20
Piperacillin tazobactum	38	12

CHART 10 :ANTIMICROBIAL SUSCEPTIBILITY OF

PSEUDOMONAS AERUGINOSA

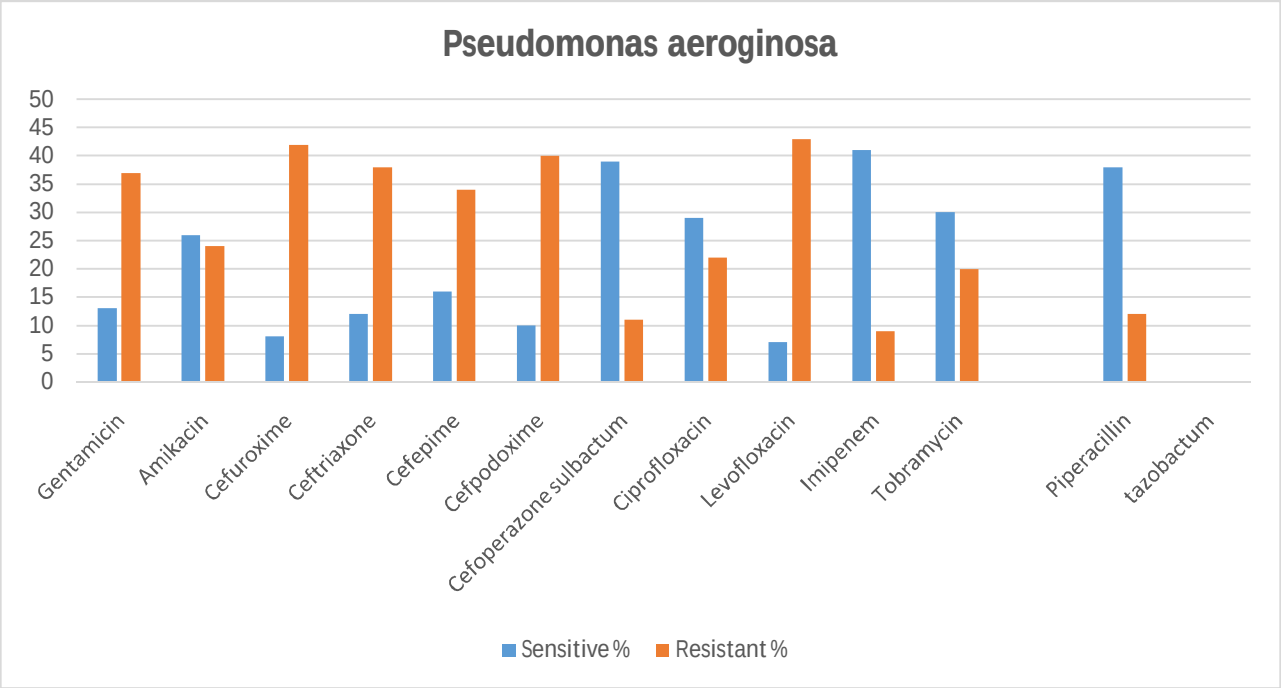


TABLE 18 : ANTIMICROBIAL SUSCEPTIBILITY OF *ESCHERICHIA COLI*

Name	Sensitive	Resistant
Gentamicin	14	36
Amikacin	37	13
Cefuroxime	13	37
Ceftriaxone	13	37
Cefepime	36	14
Cefpodoxime	0	50
Cefoperazonesulbactum	50	0
Ciprofloxacin	20	30
Levofloxacin	14	36
Cotrimoxazole	0	50
Chloramphenicol	37	13
Imipenem	50	0
Ampicillinsulbactum	9	41
Amoxicillinclavulanicacid	9	41
Piperacillin tazobactum	50	0

CHART 11 ANTIMICROBIAL SUSCEPTIBILITY OF *ESCHERICHIA COLI*

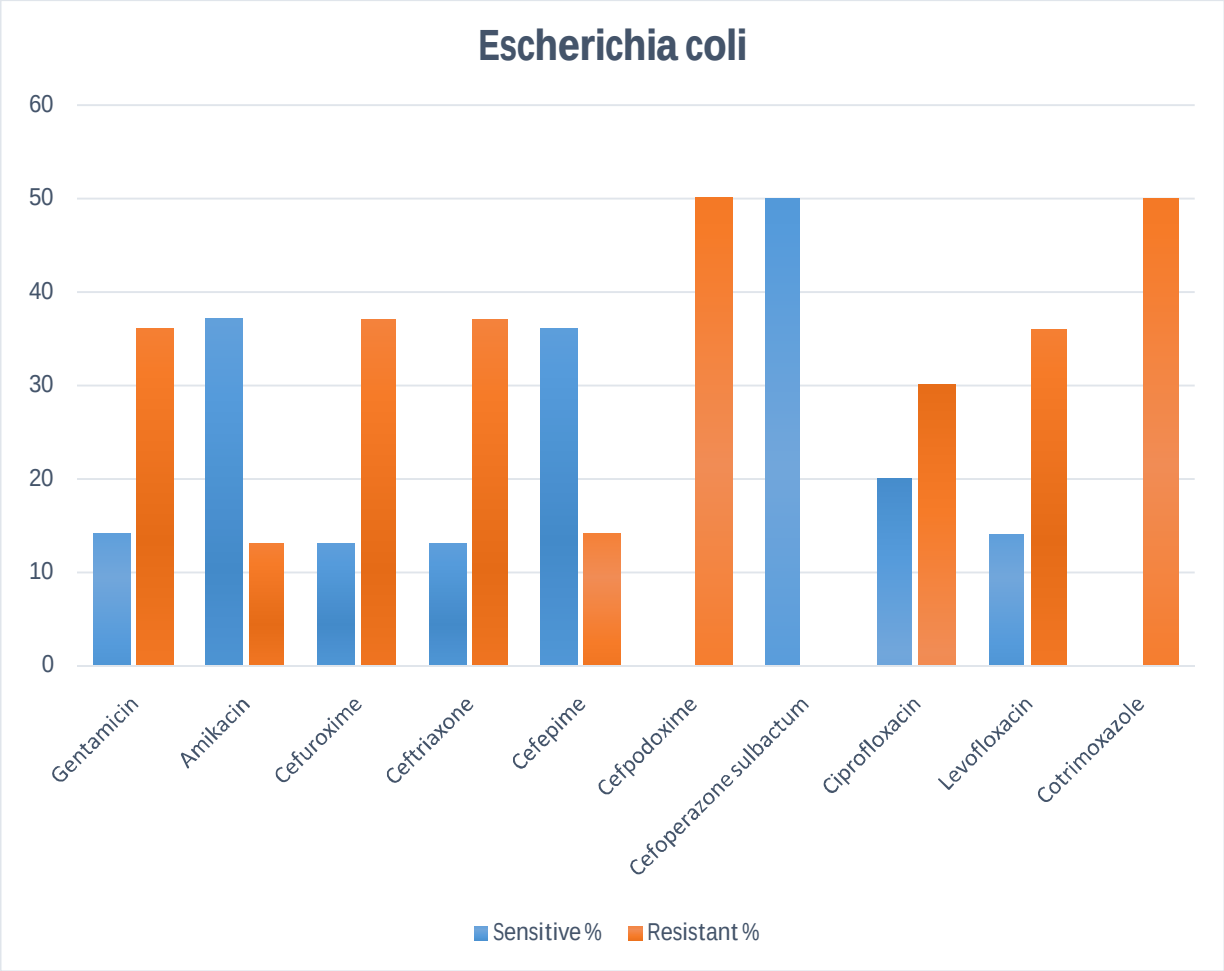


TABLE 19 :ANTIMICROBIAL SUSCEPTIBILITY OF *STAPHYLOCOCCUS AUREUS*

Name	Sensitive	Resistant
Erythromycin/azithromycin clarithromycin	13	37
cefoxitin	13	37
penicillin	13	37
cotrimoxazole	0	50
linezolid	13	37
doxycycline	37	13
tetracycline	37	13
vancomycin	50	0
chloramphenicol	37	13
Ciprofloxacin/levofloxacin ofloxacin	0	50
gentamycin	0	50
Ampicillin sulbactum	37	13
Amoxicillin clavulanicacid	0	50

CHART 12 :ANTIMICROBIAL SUSCEPTIBILITY OF *STAPHYLOCOCCUS AUREUS*

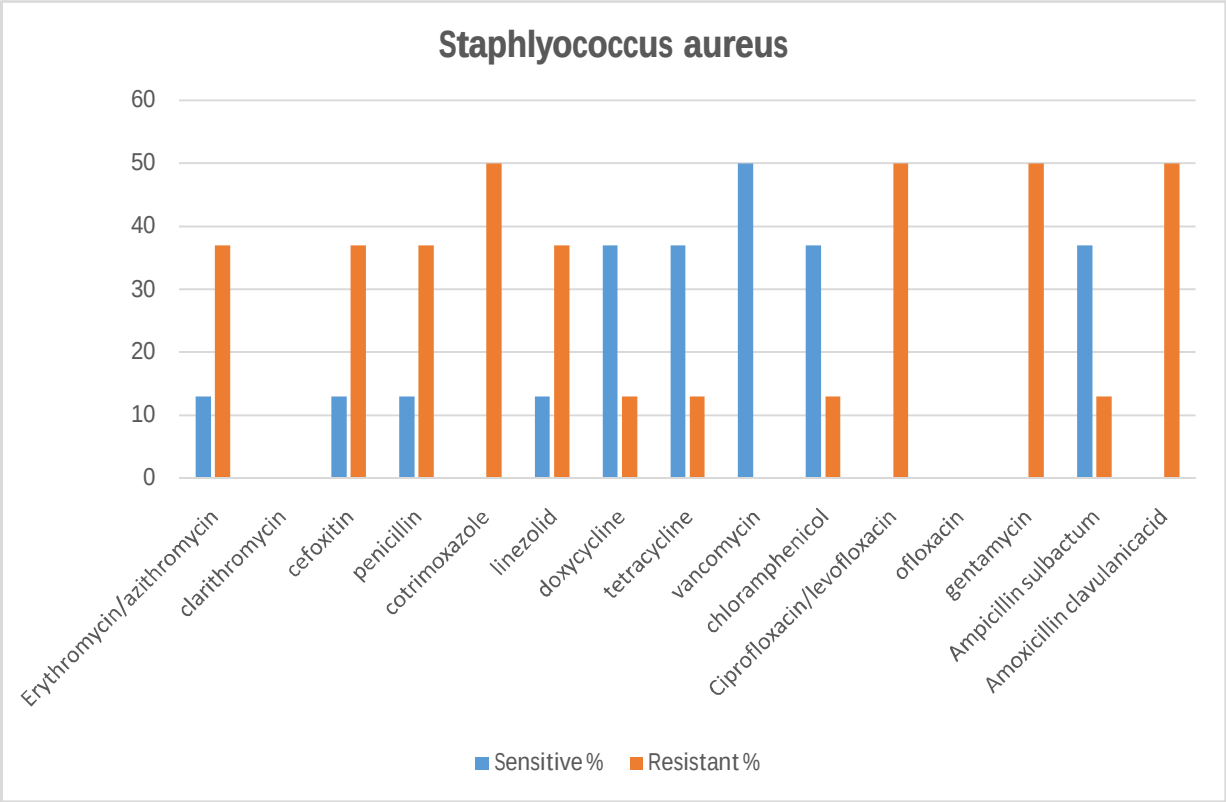
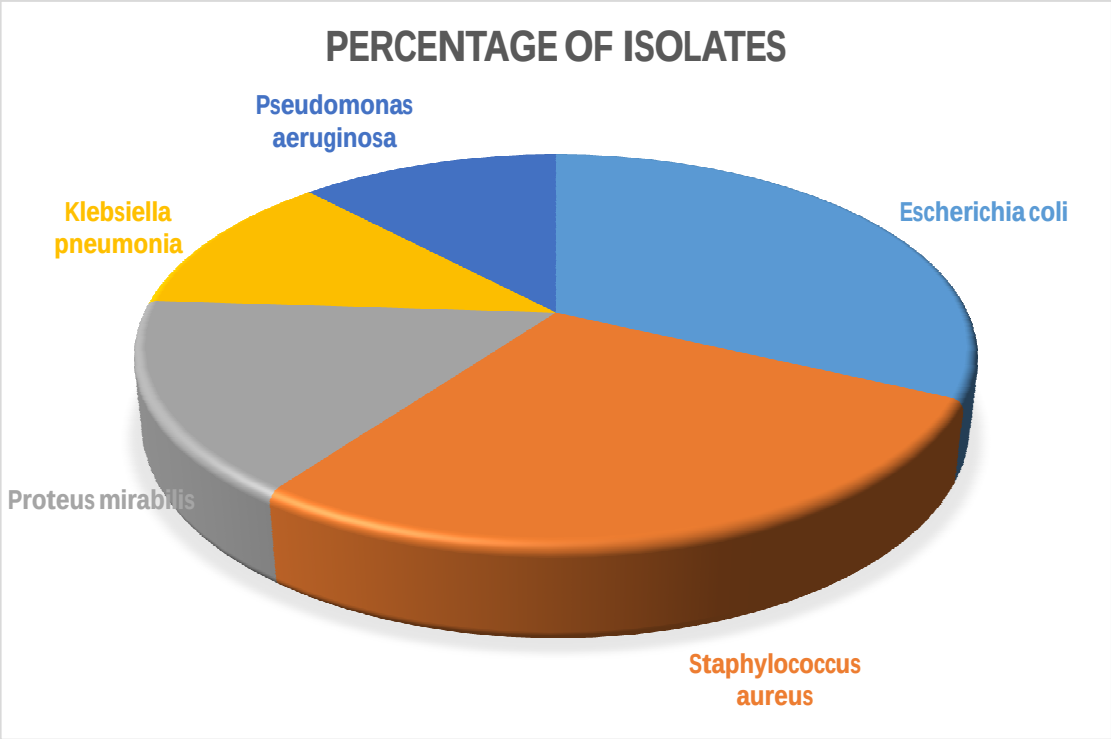


TABLE 20 :PERCENTAGE OF DIFFERENT BACTERIAL ISOLATES

Bacteria isolated from the culture	Percentage of Isolates
Escherichia coli	32
Staphylococcus aureus	28
Proteus mirabilis	16
Klebsiella pneumonia	12
Pseudomonas aeruginosa	12

CHART 13 : PERCENTAGE OF ISOLATES



DISCUSSION

Diabetes is a serious systemic disorder in which there exists high blood glucose levels. Diabetic wounds are injuries to the body tissues caused by physical trauma and proceed to deeper layers if untreated due to the high sugar level in the blood. Physicians have an important role in the prevention, early diagnosis and management of diabetic foot complications. Management however entails an extensive knowledge of the major risk factors for amputation and preventive maintenance. In the present study, we found that elderly patients of mean average age 59.2 constituted the majority of patients with foot infections. These findings are almost similar to other studies conducted where the mean age group affected was 58 years . This may be explained by the fact that foot lesions occur commonly among patients with diabetes, particularly the elderly and those with sensory neuropathy. Previous studies have shown that the susceptibility to foot infections is greater in male patients than in female patients . The present study also showed the same result with male preponderance (37 males and 13 females). Male preponderance in the present study could be explained on the basis that the males spend more time working outdoors, exposing their foot to more traumas . In the present study, we found that the majority of lesions were located on the

toes and plantar region, this is in accordance with the results of some studies .

In the study we collected 52 samples which had shown bacterial growth and 2 samples (2.94%) did not show any bacterial growth. Similar no growth samples has also been observed in other studies . Probably the cultures could have been negative because either the wounds were not infected at the time of the study or the causative agents were anaerobes. However, available evidence does not support the use of antimicrobials in uninfected diabetic foot ulcers . Most of the samples that were processed were found to contain Gram-negative bacteria 94.93%. Some studies also found that Gram-negative bacteria (*Proteus species, E. coli,* and *Pseudomonas aeruginosa*) were predominant bacteria . Increase in ratio of Gram-negative in this study may be due to immunocompromised diabetes states of the patients who were highly susceptible to hospital-acquired infections, either by colonization with environmental strains or followed invasive surgical procedures, and exert their pathogenic effects by producing endotoxin . We found that *Escherichia coli* was the most predominant pathogen isolated from the patients followed by *Staphylococcus aureus, Proteus mirabilisKlebsiella pneumoniae* and*Pseudomonas aeruginosa*. Whereas some studies stated that *Pseudomonas aeruginosa* was the most frequently one .

Source of infection, use of antibiotic drug for treatment, sample collection method, geographical variation, and type and severity of the infections can influence the diversity of pathogens in different geographical areas. All the isolates were tested for their antibiotic sensitivity and resistance pattern.

In this study, we found that *Klebsiella pneumoniae* was more sensitive to Imipenem (89.58%). It is also found similar to some researches .It is followed by Cefoperazone sulbactum (79.17%). But a research shows that *Klebsiella pneumoniae* show considerable resistant to Cefoperazone and Cefoperazone sulbactum also . Our studies had shown that Klebsiella has shown resistant to Cephalosporins. It was also supported by a study . Resistance to Cephalosporins like Cefpodoxime suggest ESBL production

Pseudomonas aeruginosa was one of the common isolate. In this study it showed a great sensitivity to Piperacillin-tazobactam and Imipenem similar to other studies . Fluoroquinolone like Levofloxacin are found to have resistance to Pseudomonas. Similar results were also shown by Lee YJ *et al.,* . In 2011, Sivanmaliappan and Sevananreported that 66.6% of *Pseudomonas aeruginosa* isolates were resistant to Gentamicin. These findings are consistent with our results (73.68%).

Next to *staphylococcus* ,*Proteus mirabilis* was the prevalent organism isolated. It is more sensitive to Imipenem. But another study also found that Proteus spp were most sensitive to Cefotaxime whereas Raja*et al.*, showed 98% sensitivity to Ceftriaxone and Patel *et al.*, showed Proteus spp were most sensitive to Cefotaxime followed by Ceftriaxone . The present study showed that 100% isolates of Proteus species were sensitive to beta lactum – lactamase inhibitor combinations such as both cefoperazone-sulbactam. It is also proved by a research . In our study we found that proteus mirabilis are resistant to Cotrimoxazole. It was also proved by some research . Fluoroquinolones are also found to be resistant. It is supported by research .

With regard to *Escherichia coli* all the isolates were sensitive to Imipenem and Cefoperazone sulbactum and resistant to Cotrimoxazole. It was partly supported by Jain Manisha . But a research found *E. coli* to be 100% sensitive to Amikacin and Cefotaxime . Resistance to Cefpodoxime as shown by most of the isolated *E. coli* suggests ESBL production. A Very Few *Acinetobacter* species were isolated. They were all found resistant to all antimicrobial discs tested.

In our study we found that *Staphylococcus* aureus isolated were 14 out of 52 isolates; of which 1 was sensitive to Penicillin and 2 were resistant. However in this study it was observed that the 2 Penicillin resistant

isolates were resistance to Cefoxitin (67%) which suggest the presence of Methicillin resistant strain. Also they are susceptible to Vancomycin. According to a cross sectional study on diabetic foot lesion carried out, the drug sensitivity results indicated that *Staphylococcus* are susceptible to Vancomycin .

The increasing incidence of multidrug resistant organisms is a potential risk factor in management of diabetic foot infections which may lead to devastating complications like systemic toxicity, gangrene formation and amputation of lower extremity.

The amputation rates in the patients is 28 percent in affected individuals of the study and remaining 72 percent responded well to antibiotic therapy there preventing amputation and help in limb salvation. The occurrence of MDRO 68 percent of the isolates and polymicrobial growth is of 48 percent among the isolates.

The multidrug resistant organisms are frequently resistant to many other classes of antibiotics also. So it is necessary for the clinician to be completely aware of the prevalence rate of multidrug resistant organisms and their management strategies.

Thus my study gives knowledge about the prevalent organisms in the hospital patients and environment. Microbial analysis can be of benefit when considered in concert with clinical observations to confirm

causative organisms. The light thrown upon by this study on antimicrobial sensitivity profile can be of essential use to the clinicians for early and appropriate attack on the bacterial flora thus controlling the infection. It facilitates the clinicians about the prompt therapy and improves the clinical outcome of the patients. Also this study emphazises the need of good infection control practices which is very important in tertiary care centre with more number of patients with a persistent risk of cross infections and health care associate infections.

This study further shows that a regular system of monitoring infection routes as well as dissemination of the data form a link between the management and health care provider. Thus this work helps in implementing and improving the strategies for better control of infections**.**

CONCLUSION

In brief, the prevalence of gram negative bacterial infection was higher in diabetic foot patients from our region. Imipenem was found to be the best drug of the choice against gram negative organisms and Linezolid for gram positive organisms. There is high prevalence of ESBL producers and MRSA which threatens us towards a more dreadful post antibiotic era. Application of molecular techniques may lead to more accurate microbial characterizations and targeted antibiotic therapy which is still farfetched in many tertiary care centres too.

Therefore, it is necessary to evaluate the different microorganisms infecting the wound on a routine basis and to know the antibiotic susceptibility patterns of the isolates from the infected wound in patients with diabetic foot lesions which ultimately reduces the rate of amputations. This knowledge is crucial for planning the treatment of these patients with the appropriate antibiotics, reducing resistance patterns, and minimizing healthcare costs.

I hope the data presented on this article can assist the clinicians in determining the multidrug-resistant pathogens in diabetic foot ulcers.

PROFORMA

Name: IP No:

Age: SL No:

Sex: Date of admission:

Occupation: Date of surgery:

Religion: Date of discharge:

PRESENTING COMPLAINTS:

Pain Abdomen:

Fever:

Vomiting:

Distension of Abdomen:

Constipation:

PAST HISTORY:

Surgeries:

Medical conditions: Diabetes / Hypertension / Tuberculosis / Asthma / Epilepsy

FAMILY HISTORY:

PERSONAL HISTORY:

Diet: Sleep:

Bowel / Bladder: Smoker / Alcoholic:

EXAMINATION GPE:

Pallor: Icterus:

Cyanosis: Clubbing:

Lymphadenopathy:

Vitals: Pulse rate: Blood Pressure: RR:

SYSTEMIC EXAMINATION:

Per Abdomen:

Cardiovascular System:

Respiratory System:

Central Nervous System:

DIAGNOSIS:

INVESTIGATIONS:

Hb %: TC: RBS:	DC:	ESR:
Blood urea: Serum creatinine:	BT:	CT:
X – ray erect abdomen: USG abdomen:	ECG:	
HIV:	HBsAg:	

Type of Diabetes:

Duration of Diabetes:

Glycemic control:

Associated Complications:

i) Retinopathy -

ii) Neuropathy -

iii) Nephropathy –

iv) Hypertension –

v) Peripheral vascular disease:

Duration & Size of Ulcer:

Duration of Hospital stay:

Clinical Outcome:

CERTIFICATE OF CONSENT

I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research and understand that I have the right to withdraw from the research at any time without in any way affecting my medical care.

Name of participant _____

Signature of Participant _____

Date _____

If illiterate

A illiterate witness must sign (if possible, this person should be selected by the participant and should have no connection to the research team).

I have witnessed the accurate reading of the consent from to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of participant _____

Signature of Participant _____

Date _____

I have accurately read or witnessed the accurate reading of the consent from to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of participant _____

Signature of Participant _____

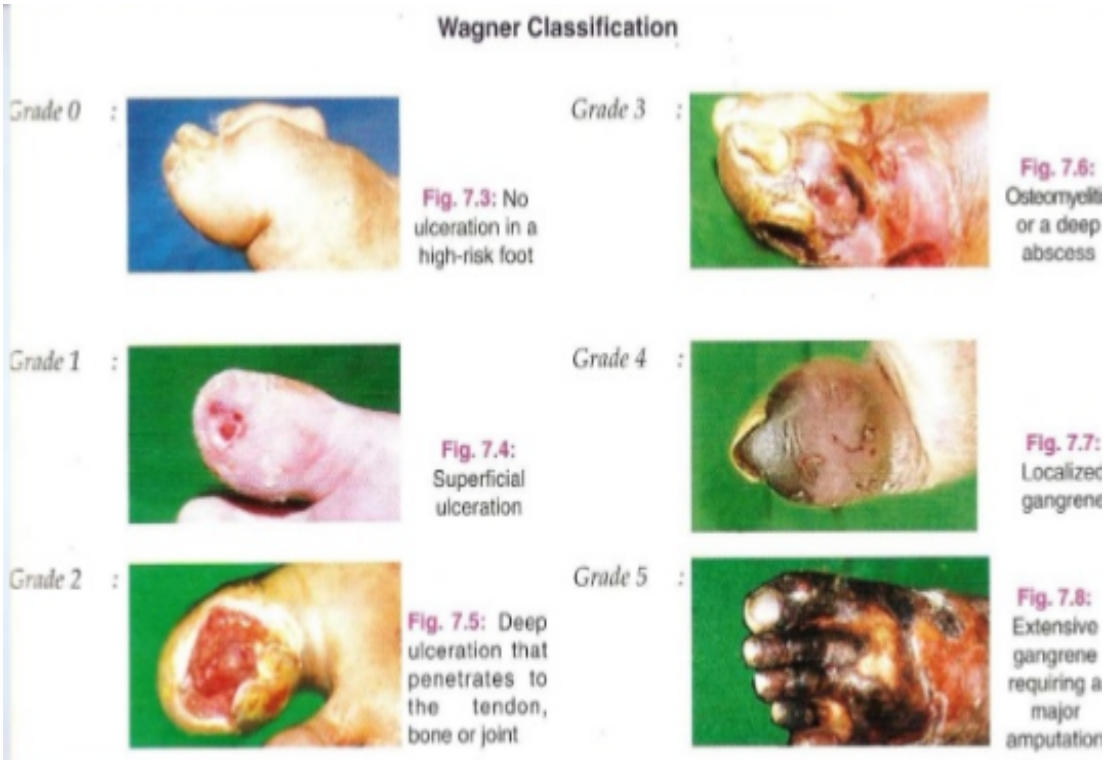
Date _____

Thumb print of participant

A copy of this Informed Consent From has been provided to participant _____ (initialled by the researcher / assistant

LIST OF ABBREVIATIONS

ATCC	-	American Type Culture Collection
BAP	-	Blood agar plate
CFU	-	Colony Forming Unit
CLSI	-	Clinical and Laboratory Standards Institute
CONS	-	Coagulase Negative Staphylococcus
DFU	-	Diabetic Foot Ulcer
DFI	-	Diabetic Foot Infection
DM	-	Diabetes Mellitus
EDTA	-	Ethylene diamine tetra acetic acid
ESBL	-	Extended Spectrum β- lactamase
GNB	-	Gram Negative Bacteria
GPC	-	Gram Positive Cocci
MIC	-	Minimum Inhibitory Concentration
MSA	-	Mannitol Salt agar
MSSA	-	Methicillin Sensitive Staphylococcus aureus
MRSA	-	Methicillin Resistant Staphylococcus aureus
NAP	-	Nutrient agar plate
PAD	-	Phenyl Alanine Deaminase
QC	-	Quality Control



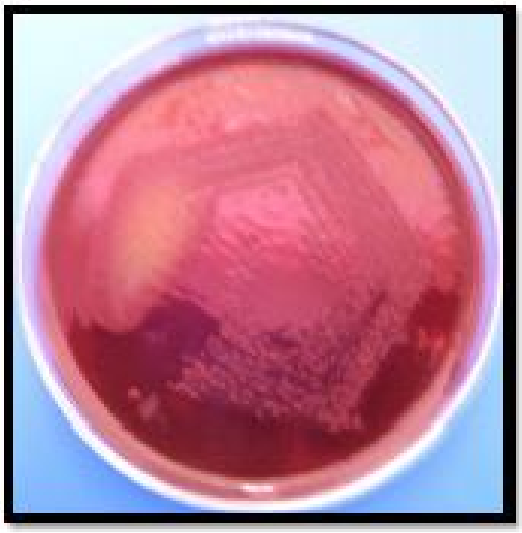
NEUROPATHIC FOOT

WOUND DEBRIDEMENT

BACTERIA IN CULTURE MEDIA

STAPH.AUREUS IN NUTRIENT AGAR

E.COLI IN MACCONKEY AGAR



PSEUDOMONAS IN NUTRIENT AGAR

KLEBSIELLA IN MACCONKEY AGAR

II) UNIVERSITY OF TEXAS FOOT ULCERATION CLASSIFICATION

STAGE	GRADE			
	0	1	2	3
A	Pre or post lesion, intact	Superficial ulcer	Penetrating to tendon or joint capsule	Penetrating to bone or joint space
B	+Infection	+Infection	+Infection	+Infection
C	+Ischemia	+Ischemia	+Ischemia	+Ischemia
D	+Infection and Ischemia	+Infection and Ischemia	+Infection and Ischemia	+Infection and Ischemia

Table : Classification of DFI : PEDIS and IDSA

Table 1-1. Classification of DFI: PEDIS and IDSA

Clinical Manifestation of Infection	PEDIS Grade	IDSA Infection Severity
No symptoms or signs of infection	1	Uninfected
Local infection (only skin and subcutaneous tissue). If erythema, must be > 0.5 cm to ≤ 2 cm around the ulcer	2	Mild
Local infection with erythema > 2 cm, or infection involving deeper tissues (e.g., abscess, osteomyelitis, septic arthritis, fasciitis) and < 2 signs of the systemic inflammatory response syndrome (SIRS)	3	Moderate
Local infection with ≥ 2 signs of SIRS: Temperature > 38°C or < 36°C, HR > 90 beats/min, RR > 20 breaths/min or $Paco_2$ < 32 mm Hg and WBC > 12 x 10^3 cells/mm^3 or < 4 x 10^3 cells/mm^3 or ≥ 10% bands	4	Severe

HR = heart rate; RR = respiratory rate.

Information from: Lipsky BA, Berendt AR, Cornia PB, et al. 2012 Infectious Diseases Society of America clinical practice guideline for the diagnosis and treatment of diabetic foot infections. Clin Infect Dis 2012;54:132-73.

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Name	AGE	SEX	DURATIO	ORGANISM	AMPUTATION	CONSERVATIVE	FBS >110	FBS>160	Amikacin	gentamycin	Ceftrixone	cefuroxime	Cefipime	cefpodoxime	Cefaperazone sulbactum	Imipenam	Levofloxacin	Amoxycilin clavulanic acid	piptaz	ciprofloxacin	ampicillin sulbacumn	chloramphenicol	tobramycin	linezolid	cotrimoxazole
noorjahan	38	F	13	e.coli		YES	YES		S	S	R	S	S		S		S	R	S	S	S	S	R	S	S
pandiyan	32	M	4	e.coli		YES	YES		S	S	R	R	S	R	S	S	S	S	S	R	S	R	S	S	S
srinivasan	36	M	8	e.coli		YES	YES		S	S	R	R	S	S	S	S	S	S	S	S	S	S	R	R	S
selvaraj	37	M	9	Stap.au		YES		YES	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	R	R
ramasamy	38	M	11	Stap.au		YES	YES		S	S	S	S	S	S	S	S	S	S	S	R	S	S	S	R	S
raman	40	M	12	Stap.au	YES			YES	S		R	S	R	S	S	S	S	S	R	S	S		S	R	S
vimala	38	F	15	Pseudo		YES	YES		R	R	R	R	S	S	R	S	R	R	S	R	R	R	S	R	S
nirmala	39	F	17	E.coli		YES	YES		S	S	R	R	R	R	S	S	S	S	S	S	R	S	R	R	R
eswari	50	F	18	Kleb.pn	YES		YES		S	S	S	R	S	S	S	S	S	S	S	S	S	S	S	R	S
jaya	53	F	19	e.coli		YES		YES	S	S	R	R	R	R	S	S	R	S	S	S	S	S	S	S	R
devi	53	F	20	e.coli		YES	YES		S	S	R	R	R	S	S	S	S	S	S	S	R	S	R	S	S
maheshwari	54	F	25	Stap.au		YES		YES	R	S	S	S	R	R	R	R	R	S	S	R	S	S	S	R	S
jeeva	42	M	13	Pseudo	YES		YES		R	S	S	S	S	R	S	S	S	S	R	S	R	S	S	R	R
jambulingam	43	M	14	Stap.au		YES	YES		R	S	S	S	S	R		S	S	S	S	R	S	S	S	R	S
prabhu	46	M	15	e.coli		YES	YES		S	S	R	R	S	S	S	S	S	S	S	S	S	S	S	R	R
arif	46	M	14	Stap.au		YES	YES		R	S	S	S	S	R	R	R	R	S	R	S	S	R	S	S	S
rajendran	47	M	14	Pseudo	YES		YES		R	S	S	S	S	S	S	S	S	S	S	R	R	S	S	R	S
kowsalya	55	F	26	Pseudo		YES		YES	S	R	R	R	S	R	R	S	S	S	R	S	S	S	R	R	S
devaraj	47	M	16	Stap.au		YES	YES		S	R	R	S	S	S	R	S	S	S	S	S	S	R	S	R	R
lakshmi	56	F	27	E.coli		YES	YES		S	S	R	S	S	S	S	S	S	S	S	R	S	R	S	R	S
vishwanathan	48	M	17	e.coli		YES		YES	S	S	R	S	S	R	S	S	S	S	R	S	S	S	S	S	S
elaiyappan	48	M	18	proteus	YES		YES		S	S	S	S	S	S	R	S	R	S	S	S	S	S	S	S	S
nataraj	48	M	19	Stap.au		YES	YES		R	S	S	S	S	S	R	S	S	R	S	S	S	S	S	R	R
mani	48	M	20	Stap.au		YES		YES	R	S	R	S	R	S	R	S	S	R	R	R	S	R	S	S	S
sasikumar	49	M	16	proteus		YES	YES		S	S	S	S	S	S	R	S	S	S	R	S	S	S	S	R	R
kannadasan	52	M	15	Kleb.pn	YES		YES		R	S	S	S	S	S	S	S	S	S	S	R	R	S	S	S	S
kulandhaivelu	54	M	18	proteus	YES			YES	S	S	S	S	S	S	R	S	S	S	S	S	S	S	S	R	S
velusami	55	M	19	Kleb.pn	YES		YES		S	S	S	S	S	S	R	S	R	S	S	S	R	S	S	S	S
selvam	56	M	20	e.coli		YES	YES		S	S	R	S	S	S	R	S	S	S	S	S	R	S	S	R	R
mohammed rafiq	56	M	20	proteus		YES	YES		S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S
saraswathi	57	F	28	E.coli		YES	YES		S	S	R	R	S	R	R	R	S	R	R	S	S	S	R	R	S
mohanraj	57	M	21	e.coli		YES	YES		S	S	R	S	R	S	R	R	S	S	S	R	R	R	R	S	S
selvi	59	F	29	e.coli		YES		YES	S	S	R	S	S	S	S	S	S	S	S	R	S	S	S	R	R
nallamuthu	58	M	22	proteus	YES		YES		S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	S	R
ramesh	58	M	23	proteus	YES		YES		R	S	S	S	S	R	S	R	S	S	R	S	S	S	S	S	S
samiyannan	60	M	24	Pseudo		YES	YES		S	S	S	S	S	S	S	R	S	S	R	R	R	S	S	R	R
ponnusamy	60	M	25	Stap.au		YES	YES		S	S	S	S	R	S	R	R	S	R	S	R	S	S	S	S	S
sakthivel	62	M	26	Pseudo	YES		YES		S	S	S	S	S	S	R	S	S	S	S	R	R	S	S	R	S
manickam	63	M	27	e.coli		YES	YES		S	S	R	S	S	R	R	S	S	S	S	S	S	S	S	S	S
krishnamoorthy	64	M	28	Kleb.pn		YES		YES	R	S	S	R	R	R	R	S	R	S	R	R	R	S	R	R	R
matheshwaran	64	M	29	Stap.au		YES	YES		S	S	R	S	R	R	S	S	S	S	S	S	S	S	S	S	R
ramathal	68	F	30	Kleb.pn		YES		YES	S	S	S	R	S	R	R	R	S	S	S	R	R	R	S	S	S
swaminathan	67	M	30	proteus	YES		YES		R	S	S	S	S	R	R	S	S	S	R	R	S	S	S	R	S
sivakumar	68	M	31	Stap.au		YES		YES	S	S	S	S	S	S	R	R	S	S	S	S	S	S	S	R	S
marimuthu	70	M	32	Stap.au	YES		YES		S	S	S	S	R	S	S	S	R	S	R	R	S	R	S	R	S
subbayian	70	M	33	Stap.au		YES	YES		S	S	S	S	S	S	S	S	S	S	S	R	S	S	S	S	S
susainathan	74	M	34	e.coli		YES		YES	S	S	R	R	S	R	S	R	S	S	R	R	S	S	R	R	R
muthusamy	74	M	35	proteus	YES		YES		S	S	S	S	S	S	S	S	S	S	S	R	S	S	S	R	S
jaganathan	74	M	36	e.coli		YES	YES		S	S	R	R	S	R	S	R	S	S	S	R	S	S	S	S	S
kittan	78	M	35	Kleb.pn		YES		YES	R	S	S	R	S	S	R	S	S	S	S	R	S	S	S	S	S

NAME	AGE	SEX	NEUROPATHY	NEPHROPATHY	RETINOPATHY	PVD	HYPERTENSION	
Jaya	53	F	+		+		+	
Vishwanathan	48	M	+			+	+	
Susainathan	74	M	+	+	+	+	+	
Pandiyan	32	M	+		+			
Manickam	63	M		+	+	+	+	
Prabhu	46	M			+		+	
Devi	53	F	+		+	+		
Selvi	59	F	+	+	+	+		
Saraswathi	57	F		+			+	
Lakshmi	56	F	+		+	+	+	
Noorjahan	38	F		+				
Nirmala	39	F	+		+	+	+	
Jaganathan	74	F	+		+			
Selvam	56	M	+	+		+	+	
Srinivasan	36	M	+	+			+	
Mohanraj	57	M			+	+	+	
Krishnamoorthy	64	M		+		+	+	
Eswari	50	F	+		+	+	+	
Kannadasan	52	M		+			+	
Ramathal	68	F	+		+	+	+	
Velusami	55	M	+	+	+		+	
Kittan	78	M	+		+	+	+	
Elaiyappan	48	M	+	+		+		
Ramesh	58	M		+	+		+	
Mohammed Rafiq	56	F			+			
Muthusamy	74	M	+	+		+	+	
Nallamuthu	58	M	+		+			
Sasikumar	49	M	+	+		+	+	
Swaminathan	67	M	+	+	+		+	
Kulandhaivelu	54	M			+	+		
Vimala	38	F	+			+	+	
Sakthivel	62	M	+	+	+			
Kowsalya	55	F	+			+		
Rajendran	47	M	+		+	+	+	
Samiyannan	60	M		+	+			
Jeeva	42	M	+	+	+	+	+	
Selvaraj	37	M	+				+	
Mani	48	M	+	+	+	+		
Devaraj	47	M	+	+			+	
Raman	40	M	+		+	+		
Matheshwaran	64	M	+	+		+	+	
Maheshwari	54	F	+	+	+			
Sivakumar	68	M	+		+	+	+	
Nataraj	48	M	+	+			+	
Marimuthu	70	M	+			+	+	
Arif	46	M	+	+	+		+	
Ramasamy	38	M	+		+	+	+	
Ponnusamy	60	F	+		+		+	
Jambulingam	43	M	+	+	+		+	
Subbayian	70	F	+	+	+	+	+	